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DETACHMENT OF THE RETINA

TREATMENT WITH MULTIPLE DIATHERMIC PUNCTURE AND ITS RESULTS

K. ŠAFÁŘ, M.D.
VIENNA, AUSTRIA

It is to the everlasting credit of Gonin that he recognized the importance of the retinal tear in the development of retinal detachment and showed the curability of this condition by operative closure. Permanent healing of retinal detachment was formerly rare and could be regarded only as accidental. Only since the time of Gonin has operative treatment of retinal detachment attained its aim, which consists in closure of the tear; in this way, it has become a rational therapeutic method. However, the instrument which Gonin used—the glowing iron—has many disadvantages. It is difficult to hit the tear with this instrument, and if several holes are present or if the tears are large, it is often impossible to accomplish closure in one operation. Postoperative hemorrhage into the vitreous body is no rarity and may spoil a successful operation. Distortion of the retina, with new tears about the place operated on, is a not unusual consequence. To avoid the disadvantages of ignipuncture, other procedures have been suggested; their aim is to close the tear and its immediate territory by producing adhesive choroiditis after cauterization of the choroid. Thus, Guist made many openings in the sclera with the trephine in the territory of the tear and applied the caustic stick. The excess potassium hydroxide was neutralized with diluted acetic acid; then the cauterized choroid was bluntly severed at the site of the trephining, and the subretinal liquid was allowed to escape so that the retina came in contact with the choroid and adhered to it.

By a number of trephinings, combined with chemical cauterization, closure of even large holes can be accomplished, and if the punctures are arranged in the shape of an arch converging toward the ora serrata (Lindner), large areas of the retinal periphery may be demarcated. This method accomplishes reattachment in cases in which Gonin's ignipuncture cannot be applied. However, it is a long, complicated and troublesome method and is dangerous in the presence of the slightest

Figures 1, 2, 5 and 6 *B* and the second, third and fourth drawings in figure 4 are from Abhandlungen aus der Augenheilkunde und ihren Grenzgebieten, Heft 16 (Beiheft zur Ztschr. f. Augenh.), Berlin, S. Karger, 1933.

deviation in its regular course. There are reports of complications involving the cornea, while extensive adhesions of the eyeball, with considerable restriction of motion, and even severer complications have been observed. To avoid the necessity of many tedious trephinings, as well as the subsequent bad effects of concentrated potassium hydroxide, which cannot always be prevented in spite of neutralization with acetic acid, Lindner makes only a few trephinings and separates the choroid from the sclera with a spatula. He then injects a dilute solution of potassium hydroxide between the sclera and the choroid; the subretinal liquid is finally drained off by perforation of the choroid. With this procedure, called the undermining method (*Unterminierungsverfahren*), peripheral demarcation can be performed, and holes, even in the macula corneae, can be made to close. If the choroid is prematurely perforated, however, the caustic liquid may get into the retina and the vitreous body and cause undesirable damage. Secondary operations may become necessary with this method which would be troublesome and dangerous because of the tight adhesions of the retina and the choroid; the spatula is liable to penetrate into the vitreous body and thus form a wrong path for the injected liquid.

METHOD

My method consists of diathermic treatment which, with the animal experiments, dates back to the spring of 1930 and is therefore chronologically related to Gonin's method. The technic is uncomplicated, simple and quick, with less trauma to the eye, and a large area may be operated on in one stage without injury to the tissues, if large, multiple or concealed tears make it necessary. For this purpose, instead of Gonin's ignipuncture, multiple punctures of the sclera are made with diathermic needles in the area surrounding the tear, which cause coagulation of the underlying choroid. After removal of the subretinal fluid through the punctures, the retina comes in contact with the choroid, which reacts to the coagulation with an adhesive choroiditis, the proliferating layer of pigmented epithelium playing a large part in the formation of the chorioretinal adhesions which seal the retinal tear so that no more vitreous can pass under the retina to lift it up from the choroid. Thus the retinal detachment is healed. The punctures are made with electrodes of various shapes which are supplied with needles 1.8 mm. long.[1]

(*A*) *Peripheral Tears.*—When there are peripheral tears or multiple holes or when an invisible tear is suspected in the periphery, delimitation of the zone of the tear is made over the healthy, although detached, retina by making an arch of punctures from ora serrata to ora serrata behind the territory of the tear or holes (according to the Staargard-Lindner method). To spare the retina as much as possible and to avoid the secondary formation of holes in the retina, this arch of punctures is performed with comblike or nail-like electrodes without stalks, which penetrate the bulbar wall. These are held with forceps which are connected with the electric current. They are left in the sclera until the arch of demarcation is completed, to prevent the subretinal liquid from escaping prematurely, which would render the bulb soft and allow the retina to approach the outer wall and

1. Šafář, K.: Ber. ü. d. Versamml. d. deutsch. ophth. Gesellsch. **49**:119, 1932.

become unnecessarily injured by the following punctures. The shape and method of application of the electrodes are shown in figures 1, 2 and 3. When the arch of delimitation is completed, the needle electrodes are pulled out; adequate filtra-

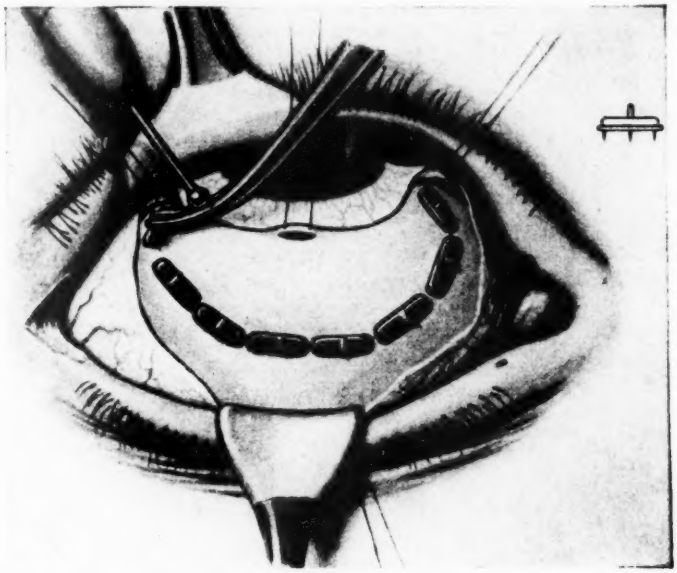

Fig. 1.—Method of treating detachment of the retina with multiple diathermic puncturing, showing one type of electrode used.

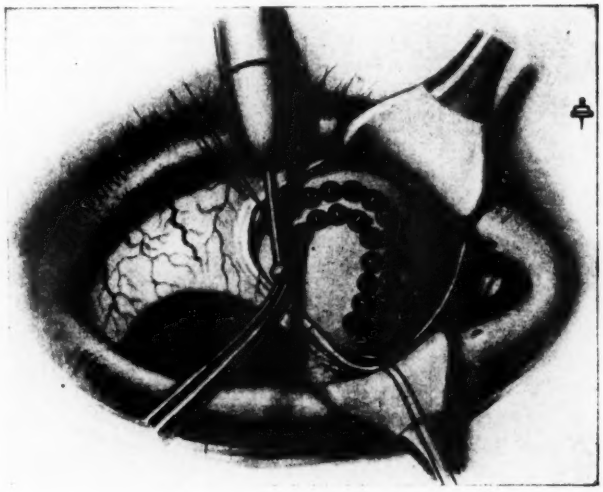

Fig. 2.—Same as figure 1, showing another type of electrode.

tion ensues through openings made by the punctures. The area peripheral to the arch as far as the ora serrata is then punctured many times with the short single needle on a stalk (fig. 4, drawing 4) to bring about flat adhesion of the retina in the territory of the tear by coagulation. Secondary holes which may have been

made in the retina disappear like the original tear if short needles are used, as a hole produced in the retina is closed with the coagulation in the choroid.

In this way chorioretinal adhesions of one quadrant or even of one half of the periphery of the fundus can be produced in one stage; also, large detachments

Fig. 3.—Electrodes and forceps in position.

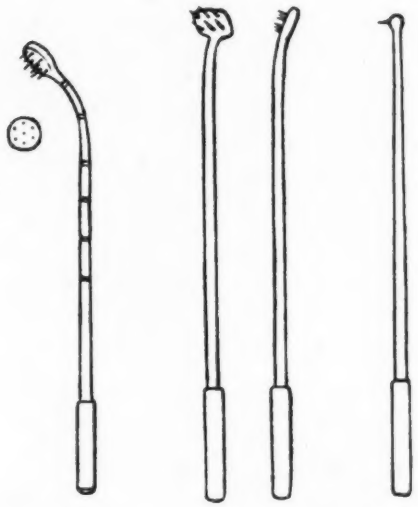

Fig. 4.—Types of needle electrodes on stalks.

of the retina from the ora serrata following contusion of the eyeball (fig. 5) can be closed in a one stage operation. The method can also be used for large rents and multiple holes in the periphery of the retina, which often occur in myopia. Retinas without visible tears may be successfully operated on in this way if the part of the periphery from which the detachment has started is soldered. The

beginning of detachment may often be deduced from the history of the first subjective symptoms, such as photopsia, flashes and the first contraction of the visual field, which appears to the patient as a black curtain. With this procedure patients can be cured who could not be treated with Gonin's ignipuncture. The sclera is exposed under local anesthesia by curved incisions of the conjunctiva of the eyeball and of Tenon's capsule, and, if necessary, with temporary resection of one or two ocular muscles. To avoid undesirable escape of current, the operative field is exposed by nonconducting retractors made of ivory or glass (figs. 1 and 2).

(*B*) *Posterior Tears.*—For tears situated posteriorly, electrodes on curved stalks are applied. These are supplied with one or several short needles which permit one or more punctures simultaneously or subsequently of the wall of the eyeball in an area corresponding to that of the tear and produce adhesive inflammation, with subsequent soldering of the retina and choroid in the territory of the tear, thereby sparing as much as possible the retina and the vitreous body (fig. 4).

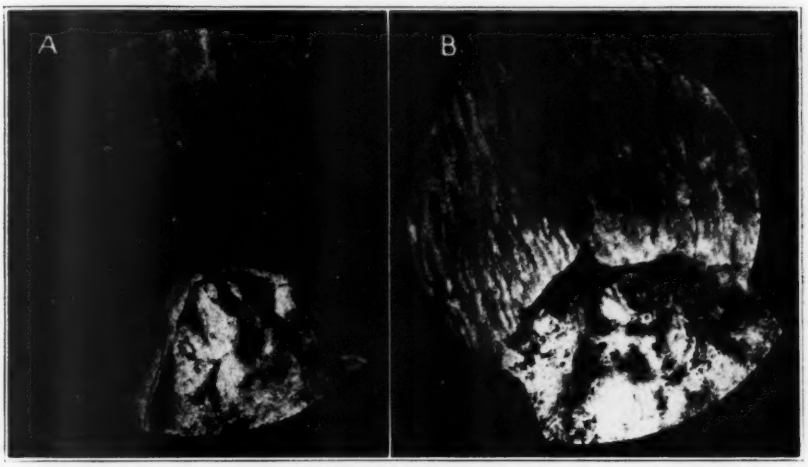

Fig. 5.—*A*, detachment of the retina after contusion, with a large tear in the ora serrata; *B*, appearance of fundus following permanent healing after one operation.

For electrocoagulation the strength of current used is: from 30 to 50 milliamperes with an electrode having one needle, from 80 to 100 milliamperes with an electrode having three needles and from 150 to 200 milliamperes with an electrode having from five to eight needles. An apparatus having this strength of current with a spark frequency of 30,000 or 50,000 allows an easy perforation of the bulbar wall, with moderate coagulation. A lower frequency is not adequate, for it does not allow sufficiently easy perforation, but causes coagulation and drying out at the scleral surface. A high frequency facilitates the puncture greatly, but the coagulation is too slight, and its inflammatory effect may be insufficient.

My diathermic method of operation, which I have developed independently of Weve and Larsson, is distinguished from the methods of these surgeons by being perforating, on the one hand, and less damaging to the retina and vitreous body, on the other.

RESULTS

In the first year of clinical use (1932), my method brought about permanent reattachment of the retina, with good vision and restoration of the visual field, in 57.5 per cent of forty unselected cases. Of uncom-

plicated cases of detachment of not more than five months' duration, which followed contusion or occurred in myopia, 90 per cent showed permanent improvement with this method. In cases of longer standing, the condition could be favorably influenced if there was not total detachment and if the retina had not shrunk; thus, reattachment could be brought about in cases of from one to three years' standing. Even cases of late detachment following retinitis proliferans in consequence of perforating injury proved to be curable with this method of extensive adhesion. Aphacic eyes rarely reacted well, and when they did so it was only in cases in which the condition had recently appeared. Postoperative complications included hemorrhage into the vitreous. Such a condition was observed in an elderly diabetic patient with an old detachment which had been treated before with ignipuncture by another oculist. This patient evidently had a hemorrhagic diathesis, since hemiplegia developed some months later. A more violent hemorrhage was observed in a myopic patient after an operation for recurrent detachment. As no tear had been visible on account of opacities of the lens and vitreous, an extremely extensive operation (involving three-quarters of the periphery of the fundus) had been performed. But in general, postoperative hemorrhages are rare, and the course of the operation is bloodless, owing to the coagulating effect of the high frequency current. Glaucoma occurred in an eye previously disposed to it, after closure of the tear, in one case; following iridectomy the detachment recurred at another site. One patient died of syphilitic sclerosis of the coronary vessels two weeks after a successful operation on the only eye with sight.

The results of this operation were still better in 1933; complete reattachment was attained in 85 per cent of the same number of eyes (forty), which has been permanent up to the time of writing. In this series, too, recent cases showed better results than old ones or those in which another method of operation had formerly been used. In old cases with a shrunken retina, reattachment by smoothing the folds and readapting the retina to the inner surface of the bulb was impossible, but in cases of partial detachment with the formation of striae, the retinas could be reattached, in spite of their having existed for months or years in that condition. Sometimes the improvement of retinal function was surprising. Postoperative complications included hemorrhage into the vitreous, which occurred in a case of old total post-inflammatory detachment; after the operation the patient got up sooner than usual and walked about, with a resulting hemorrhage. One patient who was operated on successfully died of intercurrent disease (hemorrhagic gastro-enteritis).

COMMENT

From a summary of the cases in which healing took place, the important conclusion can be drawn that early operation is necessary to cure detachment of the retina. Nowadays, when the operation is performed in time the retina can be reattached in nearly every patient who has not been operated on previously. The method of multiple diathermic punctures permits the healing of a detachment not only in the presence of large tears or multiple holes, but also when no holes can be detected. Since this method of operation is rarely followed by complications, no time should be wasted in conservative treatment, as the chance for healing and regaining useful vision quickly decreases thereby.

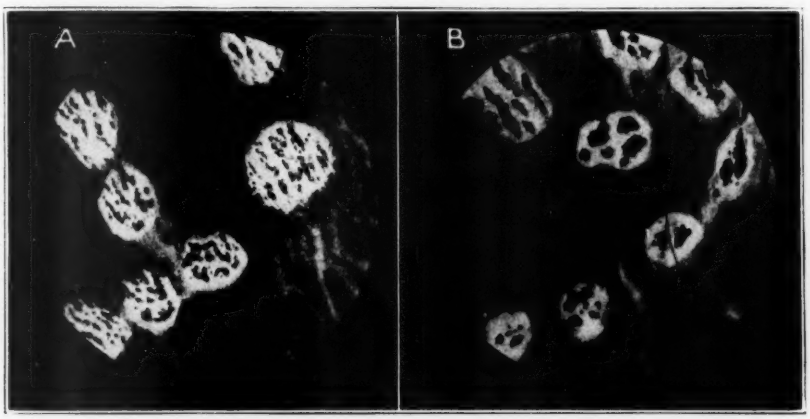

Fig. 6.—*A*, appearance of the fundus after demarcation of the territory of the hole and tear by multiple diathermic punctures. The defect of the retina can still be recognized; no occlusion of the defect followed, but there was seclusion by chorioretinal soldering extending from ora serrata to ora serrata and well posteriorly in order to close the territory of the hole from the rest of the retina. In *B*, a retinal vessel passes uninjured over an area of soldering, a sign that the internal layers of the retina were not affected.

With elderly people, attention must be paid to the general condition and absolute quiet is necessary after the operation. Both eyes should be bandaged for from ten to twelve days; then stenopeic spectacles should be worn, and the patient should rest in bed for two or three weeks. In these patients possible dangers may be avoided by such measures as deep breathing, remedies for the heart to offset the danger of pulmonary complications, moving the legs, etc. When varicose veins are present, bandaging of the legs lessens the danger of thrombosis and embolism. With younger people such counterindications are not present, and one is enabled by a comparatively simple operation to

heal nearly with certainty a disease which was regarded as incurable only a short time ago. Prerequisites for success are: careful ophthalmoscopic examination of the patient, in order to discover any tears of the retina which may be present, and an exact history, in order to deduce from the site of the first contraction of the field the beginning of the detachment, which is where the tear is probably situated—in the periphery of the retina. This is especially important in dealing with cases without a visible rent. In visible tears exact localization is to be aimed at, just as with Gonin's ignipuncture. However, this is not so important with multiple diathermic punctures, at least not in the case of peripheral tears, since with this method a larger area of the bulbar wall may be treated and the chance of hitting the site of the tear is considerably greater than with Gonin's ignipuncture. In this way one reaches the tear more easily, just as the hunter is more likely to hit a bird with shot than with a single bullet. This method can, if necessary, be used extensively with only slight intensity of current, so that injuries to the eyes can be avoided, even with punctures over a widespread area. Relapses caused by cicatricial contraction hardly need be feared; the scars produced by small needles and a low current are slight and have no tendency to shrink, as they are choroidal and do not cause proliferating retinitis.

Figures 5 and 6 show the fundus of the eye after operation with the stippling method. The delicacy of the scars is distinctly seen. They have the appearance of old choroidal scars without retinitis proliferans.

SUMMARY

With special short needle electrodes, multiple perforating diathermic punctures are made in the area of the bulbar wall where the rent of the retina is situated or is supposed to be. After the subretinal liquid has escaped through these punctures, the retina attaches to the coagulated choroid and becomes adherent to it; the tear disappears, and the detachment of the retina heals.

Since this method can be applied over a large area, with slight intensity of current, it is applicable in cases of large tears or multiple holes, as well as in those without visible tears. Location of the tears is considerably facilitated by scattering the punctures. The method is simple and efficacious, and there is little tax on the eye, particularly if the condition is recent; patients with such a condition may be operated on with nearly absolute certainty of success. Complications are rare.

Reattachment of the retina has been permanent to the time of writing in 57.5 per cent of forty eyes operated on without selection in 1932 and in 85 per cent of the first forty eyes operated on in 1933. Failures occurred in cases of old detachment, with a shrunken or contracted

retina due to an operative scar, and in old people who could not be kept flat in bed for a sufficient length of time and who were inclined to bleed; however, hemorrhages into the vitreous are rarer with this method than with other methods.

Retinas with older detachments, with the formation of striae and a boundary area, could still be reattached; sometimes there was a surprising improvement in visual function. Generally, however, early operation is indicated, for the longer the detachment persists, the less is the chance for reattachment and the regaining of useful function of the retina.

APPARENT OPTIC ATROPHY WITH RECOVERY OF NORMAL CENTRAL VISUAL ACUITY

FRANCIS HEED ADLER, M.D.
PHILADELPHIA

It is well known that visual acuity may remain normal in cases of tabetic optic atrophy for several months after the appearance of pallor of the disk. In fact, the pallor practically always precedes the diminution of central vision. It is not definitely known whether this is also true for the boundaries of the peripheral visual field, since contraction of the field is nearly always found when the pallor of the disk is first detected.

The appearance of pallor before loss of central vision has been used as an argument to prove that tabetic optic atrophy must be an ascending, and not a descending, degeneration, for in descending degeneration the central vision is always affected before the atrophy becomes evident as a pallor of the optic disk. A good example of this is the blindness which follows fracture of the orbit involving the optic foramen. In these cases the ophthalmoscope reveals a normal fundus, with an optic disk of good color and outline, and no loss of capillarity until at least from two to three weeks after the injury. Not until then can any pallor be detected. The pallor then increases rapidly until the atrophy is complete. The farther back of the eyeball the seat of the injury is, the more time it will take for descending degeneration to become noticeable in the disk.

It is also generally recognized that the degree of pallor may not correspond with the acuity of vision in that the disk may sometimes be quite white, and yet visual acuity may remain good for a short period of time. Invariably, however, such a state of affairs does not last long, and the acuity soon decreases.

A number of cases have been reported in the literature in which this discrepancy between visual acuity and pallor was a striking and practically permanent feature. Dworjetz [1] collected the reports of nine cases in which the disks were porcelain white, but the vision in one or both eyes was nearly normal. The visual fields showed some contraction, but not of marked degree.

Read before the Section on Ophthalmology of the College of Physicians of Philadelphia, Oct. 19, 1933.

1. Dworjetz, M.: Ueber die Berechtigung der Bezeichnung "Atrophia nervi optici," Klin. Monatsbl. f. Augenh. **80**:30 (Jan. 27) 1928.

Dworjetz called attention to the paradox of calling these cases complete optic atrophy from the appearance of the disks when functional tests proved that a large proportion of the nerve fibers must be undamaged. Occasionally similar cases are seen in which the optic disks are so dead white that it seems impossible for any vision to have been spared. A search of the current literature indicates that this retention of central vision, or its recovery after a temporary period of blindness, is not so uncommon in cases of poisoning with certain drugs, such as methyl alcohol, and some of the organic arsenical compounds. On the other hand, it is very uncommon for central vision to be preserved for any length of time in true tabetic optic atrophy, and when once lost the vision never returns. This point is of importance in the differential diagnosis between tabetic atrophy and atrophy due to the toxic action of arsenical preparations which may have been used in the treatment of syphilis. The following case is reported because of the recovery of central vision in spite of apparently complete atrophy of both disks. It was important in this case, from a medicolegal standpoint, to differentiate between optic atrophy caused by tryparsamide and that due to early tabes. The subsequent course of the disease brought up a number of points which were of value in making the final diagnosis between these two conditions.

REPORT OF A CASE

Tracy A., aged 38, was first seen on Dec. 11, 1929, being referred by Dr. Robert Gilman, of the Hospital of the University of Pennsylvania, for a prognosis regarding his optic atrophy. In March, 1929, injections of tryparsamide, twice a week, had been administered by a physician in the South. No tests of his vision or fields were taken at any time. After the third injection he had noticed the vision in the right eye becoming blurred, but the treatments were continued. After six treatments his vision had failed to the point of incapacitating him for work. All treatment was then stopped. At the end of a month the vision of the left eye had cleared up completely, but that of the right only partially so. In July, although he had had no further antisyphilitic treatment, the vision started to fail again, and by September he could see nothing with either eye but light.

He does not know the date of his primary infection, as he discovered that he had syphilis when a general medical examination, including a Wassermann test, was made. When first examined in my office, his vision was perception of light in each eye. The light fields were markedly contracted. The right pupil was round; the left, slightly pear-shaped. Both reacted promptly in convergence. The ocular media were clear. The disks were dead white with cleancut margins and exposure of the lamina. The arteries were reduced in caliber and were tortuous. There were no lesions of the retina or choroid. Because of the apparent complete atrophy of both disks, judged by the color of the disks and the results of functional testing, a bad prognosis was given as to the recovery of any vision.

The question immediately arose as to whether the patient's optic atrophy was due to the injections of tryparsamide or whether tabetic optic atrophy had developed and the patient was in the early stages of tabes before any involvement of

the posterior columns had become manifest. It was, of course, also possible that the tryparsamide had hastened the tabetic degeneration. Tabetic optic atrophy, as is well known, often precedes other tabetic symptoms by a considerable period of time. Optic atrophy and Argyll Robertson pupils are sometimes the initial signs of tabes when the patient is first examined. It is unusual, however, for the optic atrophy to have developed to the extent of almost total blindness without the appearance of involvement of the posterior columns, such as loss of patellar reflexes and a positive Romberg sign.

The pupils of this patient reacted sluggishly to light and were prompt in convergence, but the reduction in the sensitivity to light was so great that one could not be sure whether one was dealing with an Argyll Robertson pupil or merely with a pupil failing to react because of damaged afferent visual fibers.

In February, 1930, the light fields were distinctly larger, and the patient could discern a large letter with the left eye. He had been receiving strychnine, mainly as a placebo. He was referred to Dr. de Schweinitz, in consultation, who found the vision in the right eye limited to perception of shadows, and that of the left 1/60 (eccentric). Dr. de Schweinitz called attention to the contraction of the smaller arteries with moderate perivascular streaking along the veins and arteries, and pointed out that in addition to degeneration of the optic nerve there were signs of early arteriosclerosis. He pointed out that all of these lesions could be the result of syphilis, but he regarded the injections of tryparsamide as sharing the larger part of the responsibility. He agreed that the visual prognosis was essentially bad, and that it was more than doubtful if any useful vision could be obtained. The fact that the patient's vision varied from day to day, and from hour to hour, and that he could at times pick out large letters on the chart with his left eye indicated that something might be done to stimulate the nutrition of the optic nerve. Dr. de Schweinitz advised the use of electrical treatments as a final resort and put the instrument in his office at my disposal for this purpose.

The patient was accordingly given sinusoidal current, a binocular electrode being placed over the eyes with a pad at the nape of the neck. This was done for five minutes, once a week. The patient stated that after four treatments he noticed slight improvement in his vision, and it was noted that after a few minutes of careful fixation he could at times make out with either eye a large letter held close to him. By the following month there was no doubt that the patient's vision had improved considerably, that of the right eye being 6/100 and that of the left, 6/60. The vision gradually improved, with periods of remission, until by December, 1930, it was 6/6 in the right eye, and 6/6— in the left. No change could be detected in the appearance of the disk or the retinal vessels. There was no enlargement of the visual field, however, at any time. A test object at 10 mm. could be seen only at fixation in either eye, and the patient had to be led around the room or grope his way by carefully searching over the ground in front of him. From December, 1930, until the present time his condition has practically remained unchanged. The response of the pupils to light has improved considerably, however, and no further symptoms indicative of tabes have developed. Although the improvement in central visual acuity coincided with the electrical treatment, I do not believe that this is anything more than a coincidence.

COMMENT

The patient's statements indicate that the optic atrophy was due to the improperly controlled administration of tryparsamide, and yet when he was first seen there was the possibility either that he had a beginning

optic atrophy from tabes or that the treatment had hastened the onset of a tabetic atrophy. Optic atrophy is often the first sign of tabes, before the loss of tendon reflexes, the Romberg sign or other tabetic symptoms. In fact, the only other symptom which is likely to precede the atrophy is the development of the Argyll Robertson pupil. There were no tabetic symptoms or signs in this patient, however. His tendon reflexes were prompt, and his station good.

The course of the atrophy, on the other hand, strongly suggests the type which may follow the administration of sodium arsanilate and other arsenical derivatives. In these cases the pupillary reactions are nearly always maintained; central vision is preserved until the last, with marked contraction of the visual fields, especially on the nasal side, according to Wilbrand and Sänger. The whole disk is pale, and the arteries show considerable contraction.

There are, it is true, a few cases in the literature of undoubted tabetic optic atrophy in which the visual acuity remained fairly normal for long periods of time after the development of pallor of the disk and tabetic symptoms of the spinal cord.

Von Hippel [2] reported a case in which the better eye maintained normal acuity for seven years, although from the time the patient was first seen the papilla was dead white and the field somewhat contracted.

Wilbrand and Sänger [3] reported a case of a woman with Argyll Robertson pupils and optic atrophy. The tendon reflexes were absent, and a positive Romberg sign was elicited. The vision in the right eye was 6/6 and that of the left, 6/18. The field on the right side was contracted to within 30 degrees; that on the left to within the 10 degree circle. Eventually the sight was lost.

These authors pointed out that such cases are analogous to those of peripheral interstitial neuritis of the optic nerve, in that central vision is well preserved, the color sense is retained and the fields are concentrically contracted. They also make the statement that in those cases of optic atrophy in which the field is constricted down to fixation and central vision is affected early in the course of the disease the optic nerve is probably involved close to the eyeball. On the other hand, those cases in which the field is contracted, but central acuity remains good, speak for the situation of the disease process more posteriorly in the orbit or even in the intracranial course of the nerve. This is borne out by the case reported by Dufour and Gonin in which there was a markedly contracted field with normal central acuity in one eye, whereas there was a hemianopic defect in the visual field, with slight reduction in acuity in

2. von Hippel, E.: Jahresb. ü. d. ges. Ophth. **51**:615, 1924.

3. Wilbrand, H., and Sänger, A.: Die Neurologie des Auges, Wiesbaden, J. F. Bergmann, 1913, vol. 5, p. 550.

the other eye. Postmortem examination showed tabetic lesions in the chiasm and intracranial portion of the nerve.

At no time since the patient whose case is reported here was under my care could any hemianopic character be made out in the field defects, although the nasal field in each eye seemed to be more affected than the temporal field. But the sparing of central vision with such marked contraction of the peripheral field indicates that the lesion must be in the optic nerve in that portion where the macular fibers leave their central position. The fibers supplying the fovea must have escaped damage, and sufficient fibers must remain to conduct impulses in spite of the fact that the pallor involves the whole of the disk. This is borne out by the fact that central color vision was eventually entirely recovered.

The case demonstrates that one cannot be too dogmatic about the state of the nerve fibers from the color of the disk. Pallor of the disk may be due to one or all of the following factors: low hemoglobin content of the blood, diminution in the number of capillaries on the disk, overgrowth of glial fibers on the disk and finally actual loss of the nerve fibers. The disk may be quite pale without actually being atrophic, and vision may be temporarily lost in the presence of a pale nerve head, and yet the nerve fibers may not be actually destroyed. One must suppose that in this case the temporary loss of central vision was due to what Cushing has so aptly termed "physiologic block." When this was relieved, the function of the fibers which were not destroyed was completely recovered. It is well known that once an optic nerve fiber has been destroyed it can never regenerate.

The prognosis is probably good in this case as far as preservation of central visual acuity is concerned. It seems doubtful that there will be any further loss of vision at this time if, as I believe, the cause of the disaster was the tryparsamide. On the other hand, one cannot expect any improvement in the visual fields. This man affords a striking example of how important the peripheral field is in daily life. Although he has normal visual acuity in one eye and nearly normal acuity in the other, he has to be led around the room, and any gainful occupation is practically impossible. He offers a contrast to the person whose central acuity is reduced to 6/60 or less from a central scotoma, but whose peripheral fields are full. Such a person is unable to read but needs no assistance in getting about and can easily find an occupation in which he can be self-supporting.

CONGENITAL CYST IN THE VITREOUS

STEPHEN G. SEECH, M.D.
LOS ANGELES

According to the number of reports in the ophthalmic literature, cysts in the vitreous are rather uncommon.

The first case of a cyst in the vitreous was reported in 1899 by Tansley.[1] A 17 year old youth had a floating, spherical cyst in the vitreous, with a few spots of pigment on the posterior surface of the cyst, which were seen through the capsule.

In 1903, Troncoso [2] reported a case in a girl, aged 15, who had a floating oval cyst, with considerable pigment on its surface.

In 1913, Shine [3] presented before the Section of Ophthalmology of New York Academy of Medicine a patient with a globular, semi-transparent, grayish cyst floating freely in the vitreous. He considered it to be an epithelial outgrowth from the ciliary body.

In the same year, Brewerton [4] reported two cysts in the vitreous of a 49 year old man, which he found after treating the patient for iridocyclitis. The cysts were oval, transparent and in a fixed position.

In 1929, Scarlett [5] presented before the American Ophthalmological Society an 8 year old girl with a freely movable cyst in the vitreous, over the surface of which were sprinkled numerous black dots the size of a pinpoint. The body of the vitreous was liquefied.

In discussing Scarlett's report, Hill [6] reported the case of a middle-aged man who had atrophic optic disks and floaters in both vitreous bodies; in the right vitreous there was a floating cyst, apparently unattached, moving slowly with the movements of the eye.

In McLean's [7] case, presented in 1932 before the Baltimore Medical Society, the vitreous of the left eye showed a pea-sized globular body, movable within a limited area. McLean cited Litinsky, who reported a bilateral cyst in the vitreous in a patient with pigmentary retinitis; the cystic formations were attached to the retina by transparent fibers.

Presented before the Los Angeles Society of Ophthalmology and Otolaryngology, Nov. 27, 1933.

1. Tansley, J. O.: Tr. Am. Ophth. Soc. **9**:507, 1899.
2. Troncoso, U.: Ann. d'ocul. **30**:341, 1903.
3. Shine: Arch. Ophth. **42**:398, 1913.
4. Brewerton: Tr. Ophth. Soc. U. Kingdom **33**:93, 1914.
5. Scarlett, E. P.: Tr. Am. Ophth. Soc. **27**:154, 1929.
6. Hill, E., in discussion on Scarlett.[5]
7. McLean, A. L.: Am. J. Ophth. **16**:51 (Jan.) 1933.

Recently, Hurwitz [8] reported a freely floating, round cyst in the vitreous of a 13 year old girl. Although many irregular spots of pigment were present on the surface, the body of the cyst was sufficiently transparent to permit a clear view of the retinal blood vessels. Hurwitz felt that the cyst was congenital and that it had originated in the ciliary process and later became detached into the vitreous.

My case shows many points of resemblance to the cases cited and undoubtedly belongs to the same category.

REPORT OF A CASE

History.—A woman, American born, white, aged 23, came under my observation on Feb. 25, 1933, through the courtesy of Dr. D. N. Alcon. She complained of headaches and intermittent blurred vision of the left eye of a few seconds' duration. There was no history of trauma. No one in the family had a similar visual disturbance.

Examination.—The patient had worn glasses for ten years and had compound myopic astigmatism of both eyes. Vision of the right eye was 20/20—3 with —6 D. sph. ⌒ —2 cyl., axis 180°, and of the left eye, 20/20 with —1.25 D. sph. ⌒ —2.50 cyl., axis 180°. When the eye assumed a stationary position looking forward, a translucent, slightly oval cyst, about one and a half times the size of the disk, could be seen in the center of the lower half of the vitreous. The cyst consisted of two parts, the lower one being large and round and the upper one, small and flat. The two parts were separated by a constriction, the small part thus giving the appearance of a cap. A transparent fiber extended from the cap upward into the vitreous; the distal attachment of the transparent fiber could not be seen. The cyst showed considerable black pigment in several places, apparently on its inner surface. With the movements of the eye the cyst bounced freely in the vitreous, assuming different positions; in the end it always returned to its original place, righting itself on the transparent fiber. When the cyst became displaced in front of the disk, the contours of the disk could be made out, not without difficulty, through the transparent, nonpigmented parts of the cyst.

On slit-lamp examination, the fibrils of the anterior portion of the vitreous were found to be dissolved. There are numerous dots of brownish pigment scattered around in this part of the vitreous. The cyst itself could not be seen with the biomicroscope.

With the exception of the foregoing findings, both fundi were normal.

Different opinions were advanced to explain the origin of this cyst. Alt considered it to be a degenerated adenoma of the ciliary process, which broke loose into the vitreous. Randall believed that it was a cystic growth at the site of a coloboma, which was freed from its attachment and set loose in the vitreous by the closure of the congenital cleft. According to Troncoso, the cysts in the vitreous originate from the uvea, which accounts for the accumulation of pigment on the surface of the cyst. Litinsky gave two explanations: first, that the cyst may be due to an inflammation or a degeneration of the retina, or to choroidal changes; second, that it is a retrogressive formation of the embryonic vitreous.

8. Hurwitz, C. E.: Cyst in the Vitreous, Arch. Ophth. **9**:825 (May) 1933.

CONCLUSION

The small number of cases reported, the lack of observation extending over a long period of time and the absence of histologic studies make it difficult to arrive at a definite opinion as to the nature and origin of cysts in the vitreous. One point must be emphasized: In practically all cases of cysts in the vitreous there is not the slightest evidence of an intra-ocular inflammatory or degenerative process. Because of this, one feels inclined to consider the cyst as a developmental rather than a pathologic condition.

MUSCLE TRAINING IN FUNCTIONAL CONVERGENCE INSUFFICIENCIES

LEIGHTON F. APPLEMAN, M.D.
Attending Surgeon, Wills Hospital
PHILADELPHIA

While the importance of thorough examination for the correction of functional weakness and discomfort is clearly recognized and taught in postgraduate instruction, and while the subject may seem trite, I still feel that the study of muscular weakness and particularly of convergence insufficiencies is not as carefully followed as it should be. Moreover, the methods for its correction are so various that a certain amount of confusion exists in the minds of many as to the benefits to be obtained in the face of the time required to deal adequately with it and the difficulty of arousing sufficient willingness and persistence on the part of the patient to enable him to follow the treatment to a definite conclusion.

It has been my experience that the knowledge gained in postgraduate instruction is not carried out in clinical work, and that the possibilities for good results in the correction of convergence insufficiencies are lost in the press of other routine work.

This should not be allowed to occur, as the oculist is the one who should be best qualified to deal with this condition, but unless he is thoroughly alive to the necessity and importance of his dealing with it he will find the treatment of the condition taken up by others less well qualified. How much attention is being given to ocular muscle training in optometric schools I am unable to say, but I have been informed that in one establishment in this city patients may be seen exercising the ocular muscles at some of the modern devices employed for this purpose. The possibilities along this line are not hard to see, and it is only a matter of time until the public will be educated to expect work of this or a similar nature to combat muscular weakness. If the oculist neglects to search for and uncover such weakness he has not done justly by his patient.

In the routine examination of a patient suffering from asthenopic symptoms there are, in my opinion, three major factors to be taken into consideration always: (1) the refractive error, correction of which, in the majority of adults up to a certain age, is to be made by examination under a cycloplegic; (2) muscle imbalance, particularly hyperphoria, correction for which should be incorporated in the lenses; (3) deficiency

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Read at the meeting of the Section on Ophthalmology of the College of Physicians in Philadelphia on Jan. 18, 1934.

in convergence power, which has a very positive bearing on the patient's ability to do close work and which requires strengthening of the muscles concerned.

It is not now my purpose to discuss the first two of these factors, which are assumed to be corrected properly. The third factor I wish to deal with from the standpoint of my own experience and method of combating it.

I realize that several methods are employed to accomplish the same ends, namely, the relief of the patient, and are therefore justified. I urge on the younger men that this angle of the work be not neglected. In the hope, therefore, that my own experience may be of aid to others I am induced to bring it before you.

One is brought to a realization of convergence insufficiency when, in a study of the muscle balance, one discovers an exophoria of 1° or 2° for distance and of from 10° to 15° for reading. One then immediately tests the prism converging power, which normally should be from 18° to 25° and the relation to divergence as 3 is to 1. If the convergence power falls to 8° or 10° it may be considered functionally deficient, and the patient may be aware of this by a variety of symptoms on the continued use of the eyes at close work; this in spite of the fact that the other factors may have been corrected within a comparatively short time previously.

For the correction of muscular weakness of this sort one must resort to exercises that will restore normal vigor to the muscles involved. To do this the physician must be convinced of the value of this procedure and he must impart his conviction to the patient by making clear to him the reason for such a procedure and the purpose to be accomplished, in order that he may cooperate willingly. He is told that the muscles that turn the eyes and hold them in the reading position are weak, their power being a certain fraction of the normal power, and that in order to make them functionally normal they must be exercised for five minutes morning and evening regularly and persistently every day for a period covering three, four or even six months, and that he must report the progress at weekly intervals and receive additions of prism strength for further use. By way of illustration he may be told that if he had a weak arm to bring back to normal strength he would exercise with a certain weight for a certain length of time daily, increasing the weight at such intervals as he was able to use it without tire, until the vigor and strength of the muscles were restored to normal or even above normal. In the same way, he is told, it is proposed to increase the work of the eye muscles by making them overcome more and more resistance through increasing the strength of the prism against which they work.

Prisms of a low degree of strength, bases out, are then ordered for him, to be placed in a temporary frame for use at home, over his

own glasses, while he is seated, gazing at a candle flame 20 feet (6.0 meters) distant. He is to lower the prisms before the eyes, which will produce a double image that should fuse into one almost immediately; as soon as this happens he should raise the frames, and when the eyes have readjusted he should repeat the maneuver, keeping it up and doing it as often as possible during a period of five minutes morning and evening. At the end of one week he should report to the office and receive an order for a glass of increased strength, which he should use again for a week in the same way, and so on at weekly intervals until he can overcome a total of 40°, and sometimes more, in exercise for five minutes without effort.

When this point is reached, all exercises may be stopped, and even though he loses after a time some of this strength he still retains close to the normal, 20°, and continues his ordinary occupation without discomfort.

This may seem like a long-drawn-out procedure, but to me it is a perfectly rational one for obtaining a true increase in muscular vigor. No one would expect to build up general muscular vigor by giving high resistance for the muscles to work against without first a very gradual approach which would enable the patient to meet it without the strain of exhaustion.

If the patient shows only 6°, 8° or 10° of convergence power, he should be started with (*a*) two 1° or 2° prisms, bases out. The subsequent increases would be as follows: (*b*) two 3° prisms, bases out, for one week; (*c*) two 4° prisms, bases out, for one week; (*d*) two 5° prisms, bases out, for one week; (*e*) two 6° prisms, bases out, for one week; (*f*) one 6° and one 8° prism, bases out, for one week; (*g*) two 8° prisms, bases out, for one week; (*h*) one 8° and one 10° prism, bases out, for one week; (*i*) two 10° prisms, bases out, for one week; (*j*) one 10° and one 12° prism, bases out, for one week; (*k*) two 12° prisms, bases out, for one week; (*l*) one 12° and one 15° prism, bases out, for one week; (*m*) two 15° prisms, bases out, for one week; (*n*) one 15° and one 20° prism, bases out, for one week, and (*o*) two 20° prisms, bases out, for one week.

In the early stages of this exercise the patient may be warned that it is an up-hill job until he is able to use approximately two 10° prisms without tire; after that it will seem to go more easily. He is therefore encouraged and told to watch the results himself. This he can do by observing that for the first few days he will appreciate, after five minutes exercise, that the eyes have been working to an unusual degree, and then toward the latter end of the week there will be much less tire or possibly none; this observation may be made after each increase in strength of the prism. If, in his report at the week-end, it appears that the

previous week's exercises have not been done readily without tire, he is told that the strength will not be changed until another week has elapsed. In this way he is brought through a very trying course of treatment, but once he can see the gradual improvement in his ability to do these exercises and with that a gradual loss in the personal discomfort that he previously experienced, he will become enthusiastic in the continuance of them until the desired goal has been reached.

A word as to the duration of this improvement in strength. In my experience the exercises rarely have to be repeated and, if so, only after a long interval. Whether repetition is required depends, I believe, on the length of time of the original exercises which allows the muscles to accumulate real strength and vigor. To expect a permanent strengthening in two, three or four weeks is fallacious. Also to increase the strength rapidly may lead to permanent weakness due to overtire, with discouragement to the patient and abandonment of the attempt.

I shall relate the histories of only two cases out of a considerable number which have been under my observation for a number of years and which illustrate the train of symptoms from which these patients suffer and the results of this form of treatment:

REPORT OF CASES

CASE 1.—Miss M. M. Z., a nurse, aged 40, had worn glasses for fifteen years, the last pair for about three weeks. She complained of occipital headache extending to the frontal region and worse on reading and close work. Riding on the train or moving in a crowd made her nervous and aggravated the headache. The eyes burned and lacrimation was increased. She became sleepy on reading, but noticed no blurring, although the pain increased as soon as she attempted close work.

On Oct. 19, 1909, refraction under mydriasis showed, right, with +0.50 sph. +1.25 cyl. ax. 90°, 5/4; left, with +0.25 sph. + 1.75 cyl. ax. 90°, 5/4. To this, a +1.25 sph. was added in each eye for reading after allowing a deduction of +0.25 sph. from the correction for distance.

As she showed no hyperphoria for distance and only ½° of exophoria at this time for distance, it was believed that the correction for reading, which she had not worn before, would relieve her discomfort.

Four months later, Feb. 25, 1910, she returned with the statement that for a time she felt better, but that the same train of symptoms was returning. The correction for reading was increased to +1.50 sph. in each eye.

About three weeks later she reported that she had no headache and was very comfortable save for some redness and tire in reading. A test of the convergence power showed 12° with difficulty; divergence, 7°. Muscle training was suggested and she was given a 6° prism in each eye for exercise. Fusion, however, was difficult, and although the 6° prisms were changed to two 4° prisms, she became discouraged and gave up the attempt. I now believe she was started with prisms far too strong, which simply increased her difficulty.

She then passed from my observation for about three years, until Nov. 12, 1913, when she returned with the statement that her glasses had been changed several

times by others, the last time two months previously, and that she was suffering as before, only worse. A study of the muscle balance showed now 3° right hyperphoria and 2° exophoria for near vision; of this she accepted 2½° right hyperphoria for distance. This I incorporated with the manifest correction, as follows: right, +0.62 sph. +1.37 cyl. ax. 75° with 2½° prism, base down, yielding 5/4 vision; left, +0.50 sph. + 1.62 cyl. ax. 90°, giving 5/4 vision. To this +2.00 sph. was added in each eye for reading.

The convergence power at this time was 11°. She reported relief from the headache for several months.

On Oct. 12, 1914, almost a year later, she again presented herself complaining of renewal of the occipital headache; it came on soon after she began to use the eyes and became worse during the afternoon. With the glasses on she showed no hyperphoria but 2° of exophoria for distance, and hyperphoria of 1° and exophoria of 3° for near vision. Convergence power was 11°, sinking to 6° on exercising for a few minutes with prisms. Refraction showed insufficient change in her correction to account for her discomfort; therefore muscle exercises were urged. This advice was accepted and faithfully carried out over a period of four and a half months, by the end of which time she showed 60° of convergence power.

As a result of this treatment the patient was relieved of her headaches and rendered comfortable in her ordinary daily routine. I have seen her at intervals of from one and a half to two years since then and she has retained the vigor of the internal recti without the necessity of resorting to a repetition of the exercises. In 1924, ten years after the course described, she showed 20° of convergence power; at the last examination, made in October 1933, she still showed the same amount.

Case 2.—Miss A. L. C., a teacher, aged 33, was first seen in 1915 complaining of pain in the occiput and the nape of the neck on close work. Briefly, the muscle status was: exophoria of 1° at distance and from 5° to 6° at reading distance; left hyperphoria of 1° at distance and of 3° at reading distance; convergence power, 12°.

Refraction under mydriasis revealed low compound myopic astigmatism of 1 diopter in each eye. A correction of this was ordered, with a 1° prism, base down, in the left eye for constant use.

This patient was seen at intervals of one and a half to two years until 1921, during which time the left hyperphoria increased until she was wearing 4° of vertical prism correction. The convergence power had fallen to 8°. Following correction at this date prism exercises were instituted and continued for six months, at the end of which time she overcame 40° in exercising without trouble.

At the same time great relief was experienced in the ability to do sustained close work, and no further exercises were necessary until, in September 1932, eleven years after the first course of exercises, the convergence power had again fallen to 12° with difficulty. A second course of exercises was instituted covering a little less than four months, at the end of which time she recovered 40° of converging power and stated that she felt comfortable and worked easily, doing hard, close work.

RECAPITULATION

1. Training in convergence is of prime importance in the relief of asthenopic symptoms which have been unrelieved by correction of the refractive error and the vertical muscle imbalance. Therefore it should not be neglected.

2. Exercises should start with the eyes as nearly as possible in the position of rest. The natural desire for the fusion of images then brings about a quick, vigorous contraction of the internal recti muscles as soon as the images are displaced.

3. Exercises should be continued sufficiently long for a true increase in strength and vigor of the involved muscles to occur.

4. Increase in the strength of the prisms used should be very gradual, and the interval of change from one strength to another should not be less than one week.

5. After the confidence of the patient and his cooperation have been secured, the results obtained will be an abundant reward to the patient and a source of great satisfaction to the one who directs it.

308 South Sixteenth Street.

RACEMOSE ARTERIOVENOUS ANEURYSM OF THE RETINA

(ANEURYSMA RACEMOSUM ARTERIOVENOSUM RETINAE)

WILLIAM H. STOKES, M.D.
OMAHA

Congenital arteriovenous aneurysm of the retina has been mentioned so infrequently as to make it a rare anomaly. Yet it is one that should be given consideration because of its similarity to angiomatosis of the retina (von Hippel-Czermak-Lindau's disease). As a matter of fact, the first case of angiomatosis of the retina was erroneously published by Fuchs,[1] in 1882, under the name of arteriovenous aneurysm.

REPORT OF A CASE

Elizabeth G., aged 10 years, was examined at the dispensary of the University Hospital, and received treatment for blepharitis marginalis. Routine examination of the fundi revealed a curious condition in the left eye.

The vision and the pupillary reflexes in each eye were normal. Tests of the ocular muscle showed no imbalance. The conjunctival vessels were congested. Hertel's exophthalmometer gave a reading of 16 mm. for the right eye, and 18 mm. for the left eye. On palpation, there was no pulsation of the eyeball and the surrounding tissue. On auscultation, no vascular bruit was heard over the left eye and along the temporal region. Under homatropine cycloplegia, vision in the right eye was 20/15 with a correction of + 1.00 D. sph. + 0.25 cyl., axis 90, and in the left eye, 20/15 with a correction of + 1.00 D. sph. + 0.25 cyl., axis 75. Ophthalmoscopic examination showed the right eye to be normal.

On ophthalmoscopic examination of the left eye, the media were clear. The nerve head was round and normal in color. The lamina cribrosa was not seen; the rings were distinct, and there was no blurring of the margin of the disk. In the central portion of the nerve head the central retinal artery divided into three branches. The superior temporal artery, shortly after leaving the nerve head, and about 1 disk diameter from the nerve, suddenly twisted downward and communicated directly with a branch of the inferior temporal vein. The artery was slightly larger than normal. The most spectacular finding in the fundus was the marked increase in the size of the vein, which, after direct anastomosis with the artery, coiled upward, and then downward, to communicate with the inferior temporal vein. The latter vessel was extremely large—about three times the normal width—and drained into the nerve head. When this vein was fol-

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From the Department of Ophthalmology, University of Nebraska College of Medicine.

1. Fuchs, E.: Aneurysma arterio-venosum retinae, Arch. f. Augenh. **11**:440, 1882.

Ophthalmoscopic picture in the left eye.

lowed, it was seen to bifurcate just beyond and below the macula. The two branches were traced into the extreme periphery of the fundus. A spontaneous pulsation was not seen in the two anastomosing vessels, but when pressure was applied to the eyeball, pulsations appeared. A slight pressure caused pulsation of the vein, but deeper pressure caused pulsation of the artery as well.

Another arteriovenous anastomosis was seen above the nerve head. The superior temporal vein drained toward the disk in a normal manner and showed no enlargement in its caliber. About 1 disk diameter from the nerve head, a branch of this vein coursed temporally and showed an anastomosis with an artery. This artery maintained the usual location of the superior temporal artery but apparently emerged from the retina without showing a direct communication with the vessels of the disk. The artery had the appearance of communicating directly with the choroidal circulation. Below and at 6 o'clock there was a vein which drained the peripheral retina and coursed upward to disappear in the retina just below the first bifurcation of the immense anomalous vein already described. It is possible that the smaller vein communicated with the choroidal circulation, just as the artery did, although it may have been a branch of the inferior temporal vein. The retina showed no loss in transparency. The central area of the retina was normal. The foveal reflex was distinct. Nowhere was there any pigmentary disturbance.

The periphery of the fundus was most carefully searched for the presence of an angioma, but no tumor mass was found. There were no retinal hemorrhages or exudates. Transillumination of the globe did not show any shadow. The peripheral field was normal.

The family and personal history of the child was essentially unimportant. She had two brothers and three sisters, all of whom were examined, as were the father and mother, and no anomaly of the fundus was discovered. There was no history of injury, and the serologic and neurologic examinations gave negative results. The blood count was normal. A roentgenogram of the skull was negative.

REVIEW OF THE LITERATURE

In reviewing the available literature on this subject, I find seven cases described. These cases, herewith included in abstract form, are similar in that they are congenital. They are apparently all cases of racemose arteriovenous aneurysm, but the anastomosis between arteries and veins varies in manner and location.

Magnus,[2] in 1874, described an arteriovenous aneurysm of the retina in the right eye of a patient 29 years old. He expressed the belief that the condition was traumatic in origin, because of the history of poor vision. In going over the report of the case, one is impressed with the fact that the anomaly existed prior to the injury. The injury, no doubt, was responsible for the loss of vision, but not for the formation of the aneurysm. There was a direct arteriovenous anastomosis between the superior temporal artery and the vein. There was a similar anomaly between the inferior nasal artery and the vein.

2. Magnus, H.: Aneurysma arterio-venosum retinale, Virchows Arch. f. path. Anat. **60**:38, 1874.

Schleich [3] reported the second case in 1884, that of an 8 year old boy whose right eye was blind. There was no history of injury. There was anastomosis between the superior and the inferior temporal artery, with great enlargement of the two vessels. There was also an arteriovenous anastomosis between the superior temporal artery and the vein with great enlargement of the vein. A continuous blowing sound was heard with the stethoscope over the eyeball. There was no exophthalmos. The left eye was normal.

Gunn,[4] in the same year, described the ophthalmoscopic appearance of the fundus of an 11 year old girl. An arteriovenous communication was observed in the retina between the superior temporal artery and the inferior temporal vein. There was no enlargement of the vessel.

Seydel,[5] in 1899, described the left fundus of an 18 year old boy. (An unusually good drawing of the fundus is found in his report.) The anastomosis occurred between the superior temporal artery and the vein. The vein was about four times the diameter of a normal vein and showed many convolutions. The artery was also markedly enlarged and showed spindle-shaped aneurysmal dilatations. There was a distinct difference in color between the artery and the vein, but near the anastomosis there was a gradual transition in color from the artery to the vein. The media in the left eye were normal, and there were no pigmentary changes. Vision was 6/9. The visual field was normal. There was no history of trauma, and the condition was stationary.

In 1903, Kreutz [6] reported a case of pulsating exophthalmos, due he believed, to a racemose aneurysm of the orbit. The vessels of the retina were greatly enlarged, twisted and dilated. It could not be determined with certainty whether an anastomosis took place between the arteries and the veins on account of the great tangle of vessels. Probably this anastomosis occurred. Unfortunately, the report is not accompanied by a drawing. The patient was 28 years old, and there was no history of trauma. Vision was 20/40. The treatment consisted in ligation of the common carotid, but the operation had no effect on the picture of the fundus.

Ehlers,[7] in 1924, described an anomaly of the fundus in a 65 year old patient. In the center of the nerve head of the left eye the central artery

3. Schleich: Aneurysma arterio-venosum, aneurysma circumscriptum et varix aneurysmaticus retinae, Mitt. a. d. ophth. Klin. Tübingen **2**:202, 1884.

4. Gunn, M.: Direct Arteriovenous Communication on the Retina, Tr. Ophth. Soc. U. Kingdom **4**:156, 1884.

5. Seydel: Ein Aneurysma arterio-venosum (varix aneurysmaticus) der Netzhaut, Arch. f. Augenh. **38**:157, 1899.

6. Kreutz, A.: Ueber einen Fall von Ranken-Aneurysma der Arteria ophthalmica dextra, Wien. med. Wchnschr. **53**:1725, 1903.

7. Ehlers, H.: Aneurysma racemosum arterio-venosum retinae, Acta ophth. **2**:374, 1924.

of the retina divided into an ascending and a descending branch. The descending branch was four or five times as wide as the superior retinal artery and ran downward in large curves. In the periphery, the vessel turned abruptly and returned to the nerve head. In its first course, the vessel was distinctly an artery, but its appearance gradually changed, and on its return to the disk the vessel clearly possessed the characteristics of a vein. In this case one is not able to state with certainty where the change from artery to vein took place. The caliber of the whole of this great loop seemed everywhere to be the same. The vision and the peripheral field were normal.

Rentz,[8] in 1925, depicted another striking case of racemose arteriovenous aneurysm of the retina observed in a man 23 years old. The disturbance extended from the disk into the macular area, and the vision of the affected right eye was 5/4. There was a more or less distinct transition of individual arteries of the retina into veins of the same area. Arteries, capillaries and veins alike had about eight times the normal diameter. There was edema of the neighboring parts of the retina. The rest of the fundus showed enlargement and tortuosity of the otherwise normal vessels. The author regarded the condition as congenital.

COMMENT

Congenital racemose arteriovenous aneurysm is a vascular anomaly in which there is a direct communication between an artery and a vein with a marked dilatation of the intervening capillary system. Both artery and vein may be markedly enlarged and tortuous, the enlargement and tortuosity affecting the venous side of the circulation more than the arterial side.

This anomaly may resemble angiomatosis of the retina and must be differentiated from the latter condition.

8. Rentz: Aneurysma racemosum retinale, Arch. f. Augenh. **95**:84, 1925.

CHEMISTRY OF THE VITREOUS HUMOR

I. CHEMICAL COMPOSITION OF THE PROTEINS

ARLINGTON C. KRAUSE, M.D.
BALTIMORE

The chief proteins of the vitreous humor are residual protein, mucoid, globulin and albumin. The other proteins, such as peptones, which have been detected in the vitreous humor are small in molecular size. So far, these undetermined proteins have proved to be insignificant. The globulin and albumin are identical with the serum globulin and albumin, but the mucoid and residual protein are peculiar to the vitreous humor. Since the mucoid and residual protein are the proteins essential to the maintenance of the form of the vitreous humor, the study of their chemical composition will aid in the correlation of the physiology and pathology of the vitreous humor. It is for this reason that the chemical composition of these proteins is reported.

MATERIAL AND METHODS

The vitreous humor was removed from the eyes of an ox immediately after slaughter and chilled at once. All foreign substances, such as pigment, were carefully and completely removed mechanically. The vitreous humor was then filtered. The filtrate was diluted with 4 volumes of distilled water and slowly acidified with acetic acid until the maximum precipitation of the vitreous mucoid occurred. The precipitate was centrifugated off and washed successively with water, alcohol and ether. After drying at room temperature, the vitreous mucoid was ground. The percentage of moisture was determined on a separate sample of dried mucoid.

The residue on the filter paper contained large quantities of residual protein and small amounts of the other proteins. It was finely ground mechanically and mixed with from 4 to 5 volumes of water. Since the enzyme, papain, was previously found to hydrolyze albumin, globulin and vitreous mucoid but not the vitreous residual protein, it was added to a mixture of proteins sufficient to make a 0.1 per cent solution. After digesting overnight at 37 C., the residual protein was washed well with water until all soluble substances were removed. Subsequently the residual protein was washed with alcohol and then with ether. The percentage of water of the protein was determined in the same manner as for the mucoid. A small amount of residual protein was prepared without the use of papain. This protein possessed the same characteristics as that treated with the enzyme.

From the Wilmer Ophthalmological Institute, Johns Hopkins University and Hospital.

Analysis of the Proteins by the Van Slyke Method.[1]—Duplicate samples of about 2 Gm. of mucoid and a single sample of about 2.2 Gm. of residual protein were hydrolyzed by boiling for about thirty hours with 150 cc. of 20 per cent hydrochloric acid. The phosphotungstates of the bases were decomposed by the amyl alcohol-ether method. The distribution of nitrogen in the mucoid and residual

TABLE 1.—*Distribution of Nitrogen in the Mucoid and Vitrein of the Vitreous Humor, as Determined by the Van Slyke Method*

Nitrogen	Mucoid		Vitrein	
	Gm.	Per Cent	Gm.	Per Cent
Amide	0.0236	7.31	0.0086	5.45
Humin	0.0100	3.10	0.0018	1.14
Cystine	0.0102	3.16	0.0020	1.27
Arginine	0.0326	10.20	0.0237	15.03
Histidine	0.0248	7.70	0.0066	4.19
Lysine	0.0324	10.11	0.0112	7.10
Amino-nitrogen of filtrate	0.1760	54.62	0.0928	58.88
Nonamino-nitrogen of filtrate	0.0110	3.27	0.0112	7.10
Recovered	.....	99.47	.....	100.16

TABLE 2.—*Distribution of Nitrogen in the Mucoid and Vitrein of the Vitreous Humor, as Calculated from the Van Slyke Analysis in Terms of Percentage of the Protein*

Nitrogen	Mucoid, per Cent	Vitrein, per Cent
Amide	0.90	0.77
Humin	0.38	0.16
Basic	3.83	3.90
Nonbasic	7.16	9.33
Total	12.27	14.16

TABLE 3.—*Distribution of Basic Amino-Acids in the Mucoid and Vitrein of the Vitreous Humor* *

Amino-Acid	Mucoid, per Cent	Vitrein, per Cent
Arginine (calculated from analysis by the Van Slyke method)	3.88	6.62
Histidine (calculated from analysis by the Van Slyke method)	3.48	2.18
Lysine (calculated from analysis by the Van Slyke method)	6.51	5.26
Cystine (determined by the Folin and Marenzi method, as modified by Tompsett)	3.20	1.50
Tyrosine (determined by the method of Folin and Marenzi)	3.42	1.10
Tryptophan (determined by the method of Folin and Marenzi)	0.81	0.57

* Calculated on ash-free and water-free protein.

protein is shown in table 1. The amounts of amino-acids, as calculated from the Van Slyke and the colorimetric analyses, are given in table 3. The nitrogen partition of the proteins according to the Hausmann numbers is given in table 2.

1. (*a*) Van Slyke, D. D.: The Analysis of Protein by Determination of the Chemical Group Characteristics of the Different Amino-Acids, J. Biol. Chem. **10**: 15, 1911-1912; (*b*) Improvements in the Method for Analysis of Protein by Determination of Chemical Group Characteristics of the Different Amino-Acids, ibid. **22**:281, 1915; (*c*) Koehler, A. E.: Modification of the Van Slyke Method for Determining Arginine, ibid. **42**:267, 1920.

Estimation of Tyrosine, Tryptophan and Cystine.—The estimation of tyrosine and tryptophan was performed according to the colorimetric method of Folin and Marenzi.[2a] The cysteine and cystine of the protein were determined as cystine by this method [2b] as modified by Tompsett.[3]

COMMENT

Vitreous Mucoid.—The vitreous mucoid is a glycoprotein belonging to the class of conjugated proteins. It is an ester of a protein and a complex organic sulphuric acid, which is called mucoitin sulphuric acid. According to Levene,[4] this acid is composed of acetylaminodextrose and glycuronic acid joined to sulphuric acid. It is found in the mucoids of the vitreous humor and umbilical cord. In the future, perhaps, it may be observed to be common to primitive types of tissue. A similar acid, chondroitin sulphuric acid, is present in the mucoid of the sclera.

The nitrogen distribution of the vitreous mucoid shows that it belongs to the general class of mucoids of the ocular tissues,[4] although it is chemically different from corneal mucoid (table 1). This indicates that the protein group of the vitreous mucoid is a specific protein, although it is coupled with the same mucoitin sulphuric acid group that is in the corneal mucoid.

Residual Protein.—The micelles of the vitreous humor have frequently been called residual protein. I believe that this term is vague, since it has been applied to the fractions of mixed protein, and suggest that the term vitrein be employed for this micellar protein. The nitrogen distribution of vitrein is different from that of other proteins of ocular tissue, with the exception of the capsule of the lens.[5] The percentages of total, amide, arginine, histidine, lysine, amino and nonamino nitrogen in the vitrein and in the capsule are approximately equal. The amount of tyrosine is greater in the capsular protein than in the vitrein. The similarity in chemical composition of these two proteins suggests that the vitreous humor arises from ectodermal tissue. It is probable that the fibers of the zonula have a similar protein with the same chemical composition as the vitrein.

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2. Folin, O., and Marenzi, A. D.: (*a*) Tyrosine and Tryptophane Determinations in One-Tenth Gram of Protein, J. Biol. Chem. **83**:89, 1929; (*b*) An Improved Colorimetric Method for the Determination of Cystine in Proteins, ibid. **83**:103, 1929.

3. Tompsett, S. L.: A Note on the Determination of Cystine in Proteins by the Method of Folin and Marenzi, Biochem. J. **25**:2014, 1931.

4. Levene, P. A.: Hexosamines and Mucoproteins, New York, Longmans, Green & Co., 1925.

5. Krause, A. C.: Chemistry of the Lens: II. Composition of Beta Crystallin, Albumin (Gamma Crystallin) and Capsule, Arch. Ophth. **9**:617 (April) 1933.

Contrary to the belief of Duke-Elder [6] that it is the viscousness of the vitreous humor which prevents all the residual protein from passing through filter paper, the vitrein does not pass through filter paper. The micelles of the residual protein, or vitrein, hold the mucoid in their interstices. If the vitreous humor is finely ground, the mucoid is liberated and is easily separated from the vitrein. If the whole bovine vitreous humor is treated with trypsin, an exceedingly thin outer layer of vitrein may be carefully dissected away. This layer of vitrein possesses all the chemical properties of the vitrein which was prepared by grinding or by the enzymatic action of papain.

CONCLUSIONS

The vitreous humor contains two chemically specific proteins: mucoid and vitrein. The mucoid fills the interstices of the micelles of the vitrein, which prevent it from diffusing away. The chemical distribution of the vitrein in the vitreous humor suggests that the vitreous humor is not a strictly uniform gel and that the hyaloid membrane is not a condensation of the gel but is composed of vitrein. Vitrein is closely related chemically to the zonular fibers and the lenticular capsule.

6. Duke-Elder, W. S.: The Nature of the Vitreous Body, Brit. J. Ophth., Monograph supp. 4, 1930.

CHEMISTRY OF THE VITREOUS HUMOR

II. PROTEOLYSIS

ARLINGTON C. KRAUSE, M.D.
BALTIMORE

Proteolysis of the vitreous humor is directly associated with its liquefaction and degeneration. The proteolysis takes place in two ways: first, by autolysis, which may be defined as autodigestion or hydrolysis of the vitreous humor by the proteolytic enzymes which are contained in it, and, second, by heterolysis, which may be defined as hydrolysis of the vitreous humor by the proteolytic enzymes which are brought into it. In heterolysis, the proteolytic enzymes arise chiefly from disintegration of the white blood cells,[1] which may enter the vitreous humor by migration or in the exudates of blood serum. The thermolabile inhibitor for proteolytic enzymes, which is contained in the blood serum, is a factor in heterolysis of the vitreous portein. This inhibitor is easily overcome by abundant white cells, particularly such as occur in suppurative exudates. It tends to disappear after the serum leaves the blood vessels.[1]

Although attempts have been made to explain degenerative changes in the vitreous humor by various theories of colloid chemistry, it is evident that the result is unsatisfactory.

In this article a report is made on the primary changes in the vitreous humor which are caused by proteolytic enzymes.

EXPERIMENTAL PROCEDURE

The vitreous humor was aseptically removed from fresh bovine eyes (3 year old animals). All pigment and extraneous substances were carefully removed. Sufficient vitreous humor to make 2 liters was disintegrated by means of a mechanical stirrer. The vitreous humor was then divided into four fractions, each containing 500 cc. Sufficient toluene was added to provide bactericidal and bacteriostatic action. Each fraction was placed in a stoppered flask and immersed in a water bath maintained constantly at 37 C. The p_H of the mixture was adjusted by hydrochloric acid and sodium bicarbonate and was determined colorimetrically by the Clark and Lubs method and also electrometrically. For each experiment, the white blood cells, which were employed to produce heterolysis, were obtained from 2 liters of whole bovine blood.

From the Wilmer Ophthalmological Institute, Johns Hopkins University and Hospital.

1. Opie, E. L.: Intracellular Digestion; The Enzymes and Anti-Enzymes Concerned, Physiol. Rev. **2**:552, 1922.

It was found that at least 500 cc. of the vitreous humor was necessary to obtain sufficient nitrogen for analysis of the various proteins.

Analysis of each fraction was conducted in the following manner: The total nitrogen of 50 cc. of the vitreous brei was determined. The remaining brei was filtered to remove the vitrein (residual or micellar protein). Fifty cubic centimeters of the filtrate was used for analysis of nitrogen. The difference between the total nitrogen and the filtrate nitrogen gave the nitrogen of the vitrein. To another 50 cc. of the filtrate, 5 cc. of acetic acid was added, and the solution was diluted with sufficient water to make 200 cc. Experimentally, this procedure was found to produce the maximal precipitation of the mucoprotein for this investigation. After centrifugating off the precipitate, an aliquot of the supernatant liquid was used for the estimation of nitrogen. The mucoid nitrogen was calculated from the difference of the nitrogen values. To another 100 cc. of the filtrate 10 Gm. of solid trichloracetic acid was added. After shaking and allowing the solution to stand for one hour, sufficient water was added to make a standard volume. After filtering, 50 cc. was used to determine the peptone and nonprotein nitrogen. To another 90 cc. of the filtrate of the solution, from which the vitrein was removed, 5 cc. of two-thirds normal sulphuric acid and 5 cc. of 10 per cent sodium tungstate were added. After filtering this solution, the nonprotein nitrogen was determined on the filtrate. The

TABLE 1.—*Autolysis of the Vitreous Humor at p_H 7.5, with No White Blood Cells*

Nitrogen	Days			
	0	1	7	14
	Mg. per 100 Cc.			
Vitrein	4.4	4.4	4.3	4.0
Mucoid	12.3	12.0	12.4	12.0
Globulin, albumin	2.7	3.0	1.0	1.4
Peptone	6.0	4.7	0	0
Nonprotein	14.8	16.0	22.3	22.7

nitrogen of the peptone and of the globulin and albumin was determined by calculation from the different nitrogen values. Repeated analyses under the same conditions gave similar results.

For a control experiment, about 200 cc. of vitreous humor was heated at 98 C. for thirty minutes at a p_H of 7.4 and then incubated at 37 C. The amount of nonprotein nitrogen remained constant for fourteen days.

COMMENT

Table 1 shows the effect of autolysis of the vitreous humor at its approximately normal p_H of 7.5. The vitrein (residual or micellar protein) and mucoid were practically unchanged during the two weeks of autolysis. The globulin, albumin and peptone were decreased, but the nonprotein nitrogen was correspondingly increased. In other words, only the simple proteins were initially hydrolyzed in autolysis, at a p_H of 7.5. The globulin is generally the first of the simple proteins to be hydrolyzed in autolysis or heterolysis.

In table 2 it is indicated that the addition of white blood cells to the vitreous humor at a p_H of 7.5 produced the same effect. However, a small quantity of globulin and mucoid was slowly hydrolyzed.

A marked change was evident when the p_H was changed to 6.5, or the brei was made acid, and no white cells were present. Rapid proteolysis of globulin, albumin and peptone took place (table 3). Moderate hydrolysis of the mucoid occurred. A slower effect on the vitrein was evident.

The addition of white cells at a p_H of 6.5 hastened the proteolytic process of all proteins (table 4). In consideration of tables 2 and 4,

TABLE 2.—*Autolysis of the Vitreous Humor at p_H 7.5, with White Blood Cells*

Nitrogen	Days			
	0	1	7	14
	Mg. per 100 Cc.			
Vitrein	5.2	5.0	5.0	5.0
Mucoid	6.7	7.1	6.0	5.6
Globulin, albumin	4.7	1.2	1.0	0.8
Peptone	3.4	3.6	0	0
Nonprotein	12.0	15.6	20.1	20.9

TABLE 3.—*Autolysis of the Vitreous Humor at p_H 6.5, with No White Blood Cells*

Nitrogen	Days			
	0	4	7	15
	Mg. per 100 Cc.			
Vitrein	3.6	3.6	2.8	2.0
Mucoid	14.0	8.4	6.8	5.3
Globulin, albumin	4.8	0	0	0
Peptone	2.8	0	0	0
Nonprotein	11.6	24.7	27.4	30.1

TABLE 4.—*Autolysis of the Vitreous Humor at p_H 6.5, with White Blood Cells*

Nitrogen	Days			
	0	1	8	15
	Mg. per 100 Cc.			
Vitrein	4.8	4.8	2.6	2.0
Mucoid	7.7	4.0	3.0	2.6
Globulin, albumin	4.5	2.0	0	0
Peptone	3.0	4.3	0.4	0
Nonprotein	11.0	16.1	25.0	26.8

it must be remembered that the white blood cells also contained various proteins, which increased the proteins in the various fractions and which were hydrolyzed at the same time as the vitreous proteins.

In normal vitreous humor there probably occurs a minimal autolysis of the serum proteins, which filter into it indirectly from the blood. The evidence obtained from the study of other body tissues is that the albumin of the blood contains a proteolytic inhibitor which tends to prevent hydrolysis of protein.[1] It may be true that under experimental conditions the decrease of the inhibitory effect of the blood albumin

allows slow autolysis of the simpler proteins in the vitreous humor in sufficient time. This is known to happen in serum proteins in other parts of the body. Therefore, the autolytic effect in the normal vitreous humor, at a normal p_H and without the presence of a large number of white blood cells, has no pathologic interpretation. On the other hand, the constant presence of sufficient white blood cells in the otherwise normal vitreous humor produces slow softening, because proteolysis of the mucoid removes the protein which aids in producing the turgidity of the vitreous gel by its being held captive in the interstices of the vitrein. Another effect is the agglomeration of the vitrein micelles causing the formation of large floating vitreous opacities. Prolonged acidosis of the vitreous humor without white blood cells, however, causes liquefaction. The rate and amount of liquefaction depend mainly on two factors: the p_H of the humor and time. Slight or mild acidosis produces a correspondingly slow effect. The liquefaction is due to the destruction of the vitrein, or supporting structure, and the mucoid by autolysis. In the presence of white blood cells, increased acidity produces more rapid liquefaction. The loss of mucoid, but especially the loss of both mucoid and vitrein, may aid in decreasing the intra-ocular tension in certain ocular diseases.

From proteolysis of the vitreous proteins, simple protein derivatives, such as amino-acids and polypeptides form; these diffuse away from the vitreous humor and reach the blood indirectly. The insoluble substances, such as leucine, cholesterol and calcium soaps, which are also liberated in proteolysis, remain at times as crystals in the vitreous humor. The cholesterol and soaps may also arise from disintegration of the white blood cells, exudates and tissues adjacent to the vitreous humor.

This proteolytic process may be started by cyclitis, choroiditis, retinitis or any other disease which diminishes the nutrition of the adjacent internal ocular tissues, prevents the maintenance of the normal p_H of the vitreous humor and also causes the entrance of white blood cells. It is well known that inflammatory reactions with exudations tend to be acid. If the reaction is complicated by bacterial infection, the process is influenced by the nature of the organism. It is also to be expected that the thickening of the walls of the capillaries and the increased density of the membranes, particularly through senescence, tend to shift the hydrogen ion equilibrium toward the acid side by diminishing the exchange of acid products of metabolism and to augment any natural autolysis occurring in the vitreous humor.

Since the zonular fibers are chemically similar to the vitrein,[2] another result associated with the proteolytic process of the vitreous humor is

2. Krause, A. C.: Chemistry of the Vitreous Humor: I. Chemical Composition of the Proteins, Arch. Ophth., this issue, p. 960.

degeneration of the zonula ciliaris, which may produce tremulousness of the lens and, later, spontaneous dislocation.

CONCLUSIONS

The effect of autolysis and heterolysis of the vitreous proteins explains many of the pathologic changes of the vitreous humor. At the normal p_H, in the presence of a sufficient number of white blood cells, the vitreous humor may in time lose its turgidity (become more liquid) through the loss of mucoid. An abnormal increase in the hydrogen ion concentration of the vitreous humor may produce liquefaction, through decrease of vitrein and mucoid, which is hastened by the presence of white blood cells.

The material was obtained through Mr. R. L. Fox. The technical procedures were performed with the aid of Mr. W. Tauber

ANATOMIC ERROR IN USING BASE OF NOSE AS POINT OF REST FOR SPECTACLES

T. J. DIMITRY, M.D.
NEW ORLEANS

In this article I shall make known certain objections to the present pattern of spectacles. A knowledge of the anatomy of the nose reveals how errors were made and how they may be corrected. I do not question the artisan's diligentness to produce a frame pleasing in appearance and, furthermore, to produce a mechanism that makes it adaptable to the different types of noses, and I grant that he has accomplished what he sought to do. His want of anatomic knowledge, however, has permitted him to err. Hence, I shall describe the anatomic characteristics of the region at the base of the nose in order to acquaint him with the footing on which he has rested the newer type of frame.

One may carve in marble likenesses of the different types of human noses and think it proper to model spectacle frames on them, but it is not satisfactory to do so, for even a faultless carving is of little service in adapting frames without a knowledge of the anatomic structure beneath the skin of the base of the human nose.

Glasses may satisfy the esthetic artisan but prove unsatisfactory to the person who is to wear them, for there is a chasm of difference between the nose of man and its image in marble.

The designer of the new type of spectacles adapted his frame to the inanimate. Had he fitted them on a human being he would not have elected the point of rest as he did, because the nasojugal depression is an area of importance in vascular communication between the vessels of the eyes and those of the face and to permit the pad to rest naturally in this sulcus on the side of the nose interferes with vascular travel.

THE NEWER FRAME

The three essential features of this type of frame are the bridge, the pads and the temples. Though the shank from which the glasses are swung forward in front of the eyes and which transmits the weight to the side of the nose is of importance, it is not one of the three essential points; however, it will receive its share of attention when the frame is considered as a whole.

The bridge, designated the crest, is a fixed band which commonly rises as a crest. Though given different shapes and decorations, its purposes remain the same, i. e., to provide for the bridging of the lenses

together and to procure the proper distance between them so that the center of each lens is held in front of the center of the pupil. The crest should not be confused with the saddle bridge that was so popular a short time ago, for it does not mold to the base of the nose. However, with the assistance of the temples it is braced to the side of the nose by the shanks and the pads.

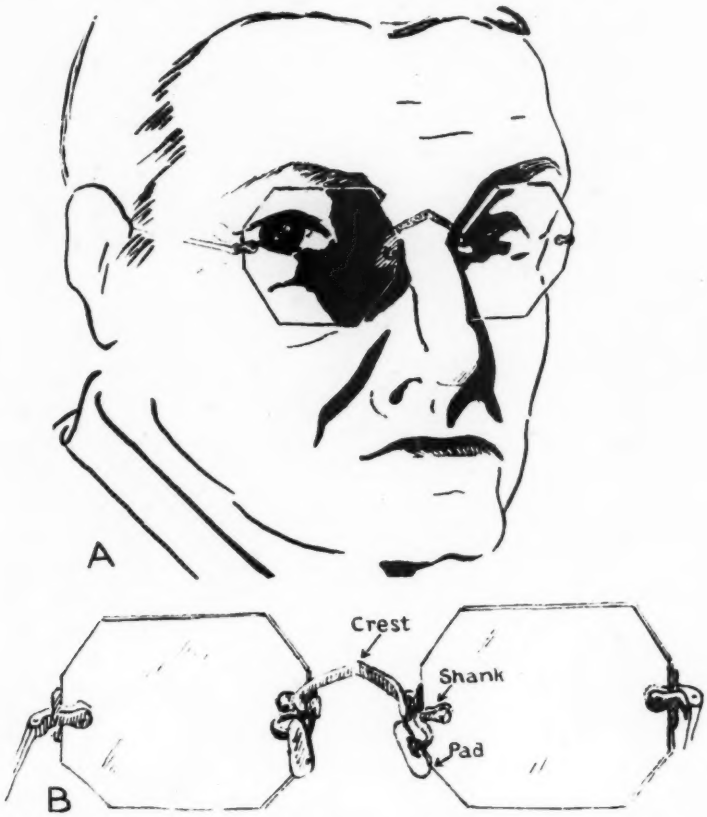


Fig. 1.—*A*, the newer type of spectacles in position, with the pads resting on the upper part of the nasojugal fold of skin; *B*, the frame, showing the crest, the shanks and the pads.

The temples provide the pressure for retaining the frames to the face, and this load, with the weight of the glass, is transmitted through the shanks and pads to the side of the nose.

The pads are usually three times as long as they are wide, e.g., 4 by 12 mm.; together with the shanks they make possible a fitting into the sulcus on the side of the nose. Their conformity to the sulcus is such that the pads rise over the ridge of the depression and readily find the bed that fulfils the esethetic ideal from a mechanical standpoint but is most objectionable when its anatomic nature is taken into consideration.

ANATOMY AT THE BASE OF THE NOSE

The framework of the base of the nose is made up of the two nasal bones and the nasal processes of the superior maxillary bones, which articulate with the frontal, lacrimal and ethmoid bones. The arch of nasal bones, though well designed to carry great weight, is further supported by the perpendicular plate of the ethmoid. The base of the nose is an example of architectural indefectibility, for the articulation of the many bones is so perfect that when one is diseased the others are responsive and sensitive. One explanation of how pressure on the bridge may provoke annoyance is had in ethmoid disease.

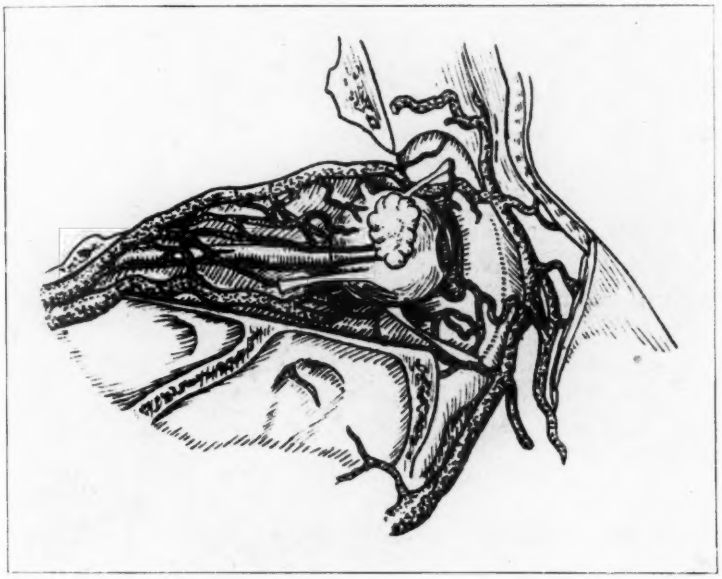


Fig. 2.—The veins of the orbit and their connections with those of the face (from Poirier).

Those familiar with craniometry will picture the area under discussion as extending from the nasion to the rhinion and laterally to the dacryon. The concavity is on the external surface of the nasal process of the superior maxillary bone, and this sulcus makes more noticeable the fold of skin which constitutes the upper part of the nasojugal fold. It is further accentuated by the attachment of the pyramidalis nasi muscle to the forward aspect of the ridge and the attachment of the orbicularis oculi and the levator labii superioris alaeque nasi muscles to the posterior portion. Immediately beneath the fold are found a few fibers of the orbicularis muscle nesting in the sulcus and the anterior facial vein and the external maxillary artery, which a short distance higher anastomose with the transverse facial vein and the infra-orbital artery. The arteries are directly continuous with the nasal branch of

the ophthalmic artery. The facial nerve and many lymphatics are also found beneath this skin fold. Moreover, there is an arborization of nerve endings, particularly branches of the infratrochlear nerve, which is given off by the nasociliary nerve where it turns to enter the anterior ethmoid foramen. This infratrochlear nerve together with the supratrochlear nerve pierces the septum orbitale for distribution to the medial canthus of the eyelid and the root of the nose, though before reaching the root of the nose, it gives off branches in the nose.

COMMENT

This study reveals the nature of the fold at the inner side of the nose, where the pad of the spectacles naturally fits. The anatomic facts are so palpable that it is unnecessary to recite how pressure on this region may provoke great annoyance by impeding the flow of blood.

Furthermore, it cannot but be realized what annoyance is caused by the nasal pad when it presses on the fibers of the orbicularis oculi muscle, whose purpose it is to open and close the lids and to participate in conveying the tears into the lacrimal sac. These muscles are excited by pressure, and this leads to repeated closure of the lids and suction of the lacrimal sac, followed by dryness and a burning sensation of the eye.

The conclusion is that spectacles of the newer type are faulty because the pad causes pressure on the upper part of the nasojugal fold, where immediately beneath the skin are vessels that anastomose to the eye and to the interior of the nose. The frame naturally rests on sulci, for it is almost impossible to retain it on the ridge or in front of the ridge.

A realization of the faults and of the reasons recited makes possible the development of a more comfortable type of frame.

FREE CYST FLOATING IN THE VITREOUS

CHARLES BRAMMAN MEDING, M.D.
Executive Surgeon, the Harlem Eye and Ear Hospital
NEW YORK

M. C., aged 42, a widow, the mother of three children, a sewing machine operator, applied for treatment at the clinic of the Harlem Eye and Ear Hospital in August, 1933, complaining that for seven months "a black ball" passed before her right eye and more and more interfered with her work. She said that it did not affect her vision for distance. There were no other symptoms. Her history in general and in relation to any specific disease was negative. Vision was 20/20 in both eyes. The conjunctiva, cornea and iris were normal. With mydriasis the lens was clear and both fundi were normal; on sudden movement of the right eyeball a round lens-shaped body immediately posterior to the lens floated across the enlarged pupil and sank slowly out of sight. Except for smudges of dark brown around its edges the body was translucent and behaved very much like a dislocated lens. A diagnosis of cyst in the vitreous was made. The slit light directed tangentially illumined the body beautifully, giving it the appearance of a gilded ball, but afforded no penetration. Examined by the retinoscope the brown smudges proved to be granular deposits of pigment on the surface, the translucent content permitting a veiled vision of the fundus to the observer and a like vision of near objects to the patient. The patient noted that the "ball" had become blacker and that it remained in the line of vision more constantly during the morning.

During numerous examinations the excursions of the cyst were found to be up and down, from side to side (sometimes filling the pupillary space), and around and around, its edges showing as silver crescents from behind the iris. It also revolved on itself.

Varying positions of the patient's head and body, recumbent or erect, did not visibly affect the position of the cyst so long as the eye was fixed, hence the possibility of examination with the slit lamp, but its response to movements of the eyeball were instant and independent of the movements of the head or body. Although no connecting band or filament could be demonstrated and its spheroidal lenslike contour did not change, the cyst seemed to be confined to a pathway or, better, to a circumscribed shallow area, all of which suggested that this body had existed from birth or at least for an indeterminate time and was as transparent as the vitreous up to the date of its interference with vision; or it may have

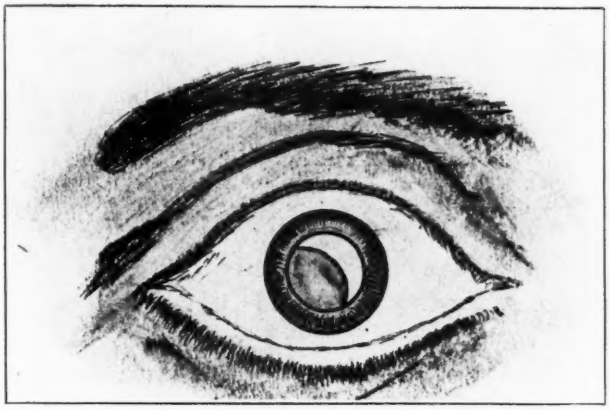
Cyst of Vitreous (Kyste flottant dans le vitre), Epithelzyste im Glaskorper

Case	Surgeon	Sex	Age	Error of Refraction	Fundus	Shape	Color	Size	Movement	Attachment	Pigment	Origin	Year	Literature
1	Tansley.....	M	17	Error	?	Round	.....	Disk size	Free	.....	P	Ciliary 3 processes	1899	Tr. Am.Ophth. Soc. **8**: 507, 1899 (case 1)
2	Koller.....	M	32	Error	?	Egg-shaped	Sea green	Three processes	.....	?	P	Hyaloid	1901	Tr. Am. Ophth. Soc. **9**: 380, 1901
3	Troncoso...	F	15	Myopia	?	.....	.....	.....	Free	.....	P	Uvea	1903	Ann. d'oeul. **130**: 341, 1903
4	Brewerton..	M	49	.....	Iridocyclitis, retinal detachment	Egg-shaped	.....	.....	None	A	No P	.....	1913	Tr. Ophth. Soc. U. Kingdom **33**: 93, 1913
5	Shine.....	F	28	Hyperopia, astigmatism	?	Globular	Steel	Twice disk size	Free	A, invisible	..	Ciliary	1913	Acta ophth. **42**: 398, 1913 (case mentioned by P. Furdenberg in discussion)
6	Scarlett.....	F	8	Error	?	Dumb-bell-shaped	.....	One half disk size	Free	.....	..	.....	1929	Tr. Am. Ophth. Soc. **27**: 154, 1929
7	Hill.....	M	45	Error	Optic atrophy	Globular	.....	.....	Free	.....	..	.....	1929	Tr. Am. Ophth. Soc. **27**: 156, 1929 (case mentioned in discussion)
8	Lavery.....	M	?	No error	?	.....	.....	.....	.....	A	..	.....	1932	Tr. Ophth. Soc. U. Kingdom **52**: 578, 1932
9	Hurwitz.....	F	13	Myopia	?	Round	.....	Disk-size	Free	.....	..	.....	1933	Acta ophth. **27**: 825, 1933
10	Meding.....	F	42	20/20 no error	Normal	Spheroid	Translucent	Lens-size	Free	No A	P	.....	1933	

escaped from or broken some attachment at that time. If it was of hyaloid origin there should have been signs in the lens or elsewhere.

The scarcity of reported cases speaks for rarity of occurrence but my experience with this case shows how easily such cysts may escape detection.

Although the cysts are interesting at present chiefly as ocular curiosities not calling for treatment, I give a tabulation of those which have been reported to date. They have occurred in both sexes,



Free cyst floating in the vitreous.

ages 8 to 49; two of the eleven persons had normal vision. A normal fundus was reported in one. It is questionable whether the cysts observed by Brewerton, Lavery and Koller should be classed with the floating, freely moving and originally transparent cysts, since they were immovable and associated with pathologic conditions. Would it not be more scientific if those connected in any way with other parts or with inflammations of the various ocular tissues were named after the affected part or considered as products of the disease? The present case and cases 1, 3, 4, 6, 7 and 9 are cases of floating cysts in vitreous, possibly congenital.

THE WILMER IRIDOCAPSULECTOMY

BENJAMIN RONES, M.D.
BALTIMORE

Occluded pupil can occur with the lens present or by the contraction of the iris following removal of the lens. The first type is most frequently seen as the result of intra-ocular inflammations with the formation of adhesions and cyclitic membranes, which cause occlusion or displacement of the pupil because of the formation of scar tissue and contraction. It also results from a traumatic iritis or cyclitis produced by a perforating injury. The second type is seen comparatively frequently following operations for cataract, especially in cases in which an iridectomy has been performed and the pillars of the iris become caught in the cataract incision. The method of procedure in these two types varies, for if the iris tissue alone is involved, an opening in it can be made by comparatively simple measures. If, however, the partially absorbed lens or a cyclitic membrane is present behind the iris, a more radical method of securing an opening must be adopted.

The restoration of a pupillary opening to improve vision has intrigued ocular surgeons for over two centuries, and the procedures for it are numerous. No single method is applicable to all cases, and there are many factors to be considered before any method can be decided on. Each patient must be carefully examined and studied from many angles. The condition of the cornea is of great importance, for its transparency is essential to the success of any procedure. If this structure is not in a healthy state, operative manipulation may lead to degenerative changes, with vascularization or the formation of opacities, and thus negate the desired result. The greatest importance is also attached to the condition of the iris. The stretching of the healthy iris over the pupillary opening makes this tissue favorable for a simple incision placed transversely to the lines of tension, for the factor of pull will then cause the slit to gape and become a large opening. The atrophy of the iris, its vascularization and its adhesion to posterior lens or capsular masses resulting from inflammations abolish the elasticity inherent in the iris, so that the important factor of gaping cannot be depended on, and it will then be necessary to cut and excise sectors of the tissue. The vascularization of the iris tissue should be studied most carefully under high magnification, and

From the Wilmer Ophthalmological Institute, Johns Hopkins Hospital.

precaution taken to avoid the most vascularized portion; such eyes are diseased, and resultant hemorrhage is not reabsorbed as easily as in healthy eyes, which leads to reorganization of the hemorrhage and closure of the opening by the scar tissue. The depth of the anterior chamber should also be noted, since this is the region in which the manipulation of the instruments occurs. Careful attention must also be given to the deeper ocular structures. If perception and projection of light are poor, it is obviously useless to believe that a newly formed opening will be beneficial, since the retina is undoubtedly diseased. The tension of the eye is another important guiding sign. Eyes with subnormal tension should be given an unfavorable prognosis, though the diminished tension is not an absolute contraindication to the procedure. An increased intra-ocular tension, on the other hand, may result in an expulsive loss of vitreous or severe hemorrhage, with collapse of the globe. It is also of the utmost importance to emphasize the fact that no operative measure should be attempted in an eye still showing signs of active inflammation, for it will only lead to an activation of the inflammation with closure of the opening and possibly loss of the eye. The slit lamp is of the greatest aid in this matter, especially in noting an increase in the visibility of the aqueous ray. No operation should be attempted in cases of sympathetic ophthalmia until it is absolutely certain that residuary inflammation is not present.

The first attempt to form a pupil by operation was carried out by Cheselden [1] (1728), who introduced the knife into the posterior chamber from the temporal side of the limbus and then pushed it forward through the iris, cutting it from behind. Sharp's [2] procedure was essentially the same, but Heuermann [3] modified it by introducing his lance through the cornea and cutting the iris from the front. Guérin [4] was the first to make a corneal section below with the broad cataract knife; through this opening he introduced a sharp iris knife with which he cut transversely through the iris. Janin [5] followed practically the same method except that he used scissors to incise the iris through the corneal incision. Various modifications of these procedures were used by Beer,[6]

1. Cheselden: Phil. Tr., London **35**:451, 1727-1728.

2. Sharp, W.: A Treatise on the Operations of Surgery, London, G. Robinson, 1782, p. 169.

3. Heuermann, G.: Abhandlung der vornemsten chirurgischen Operationen, Copenhagen, F. C. Pelt, 1754-1757, vol. 2, p. 493.

4. Guérin: Traite des maladies des yeux, Lyon, V. Requilliat, 1769.

5. Janin de Combe Blanche, J.: Mémoires et observations anatomiques, physiologiques et physiques sur l'œil, Lyon, fréres Perisse, 1772.

6. Beer, G. J.: Lehre von den Augenkrankheiten, Vienna, C. F. Wappler, 1792.

Flajani,[7] Adams,[8] Weller,[9] Mackenzie [10] and von Graefe.[11] Noyes [12] used a cataract knife for cutting the cornea and iris, and then introduced blunt hooks with which he pulled out some iris tissue, which he cut off with scissors.

These methods were satisfactory in cases of occluded pupil in which the iris was drawn up and under tension, and when a simple transverse incision into the iris caused it to gape and form a sufficient pupil. However, when the iris had lost its elasticity because of previous inflammation, or when a thick capsular membrane was adherent to its under surface, a mere incision into the iris tissue was not sufficient. For this type of case, numerous operations involving the excision of iris tissue with scissors were devised. Bowman [13] made a vertical section on both sides of the cornea and then with scissors cut the iris by two incisions diverging from the lateral points and meeting in the center, thus removing a diamond-shaped portion of tissue. De Wecker [14] carried his keratome through the cornea 1 mm. from the upper limbus, and then pushed the tip of the instrument through the iris or scar tissue into the vitreous. Iris scissors were then introduced, one blade behind and one blade in front of the iris, and two convergent cuts made downward, thus removing a triangular section with the apex below. This apex could be placed just below the center or even at the lower angle of the anterior chamber. In some cases de Wecker also made his sections below and cut out a triangle of iris tissue with its apex upward. The danger of this type of operation is the loss of a large amount of vitreous due to the yawning corneal wound through which the scissors are introduced. Elschnig [15] attempted to minimize this danger by introducing a corneal knife subconjunctivally through the sclera at the temporal limbus, carrying it through the anterior chamber and bringing it out through the sclera at a corresponding point opposite. De Wecker scissors were then introduced through these lateral openings, and a triangular sector, apex downward, was cut out of the iris.

This attempt of Elschnig to decrease the hazards of the operation was not entirely successful. The advantage of his procedure lies in

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7. Flajani: J. d. Chir. u. Augenh. **3**:130, 1821.

8. Adams, W.: Practical Observations on Ectropium, Artificial Pupil and Cataract, London, J. Callow, 1812, p. 37.

9. Weller, C. H.: Ueber künstliche Pupillen und eine besondere Methode diese zu fertigen, Berlin, Schüppel, 1821.

10. Mackenzie, W.: Diseases of the Eye, London, Longmans and others, 1840.

11. von Graefe: Klin. Monatsbl. f. Augenh., 1869, p. 431.

12. Noyes: Ann. d'ocul. **66**:151, 1872.

13. Bowman: Tr. Internat. Ophth. Cong., London, 1872, p. 179.

14. de Wecker, L.: Chirurgie oculaire, Paris, O. Doin, 1879.

15. Elschnig: Klin. Monatsbl. f. Augenh. **50**:138, 1912.

his small lateral incisions, but therein also lies its chief defect. The incisions are so small and are placed in such a manner that the manipulation of the scissors cannot be carried out with ease and speed.

The following procedure has been used for many years by Prof. W. H. Wilmer. This operation, as done by him and his resident surgeons, has yielded extremely satisfactory results, for it utilizes all of the advantages of de Wecker's operation, with the easy accessibility of the iris tissue, and adds provisions of safety which are of the greatest importance. This method has been used regularly at the Wilmer Institute for the past six years in all cases of occluded pupil resulting from ocular inflammation. Careful preoperative studies were carried out in all the cases, and if any signs of residual inflammation were present, the operative procedures were contraindicated at that time. Care was also taken to eliminate all possible sources of focal infection, such as the teeth, tonsils, sinuses and genito-urinary tract, as well as to decrease any hypersensitivity to tuberculin. The clotting time of the blood was also checked to determine whether any tendency toward hemorrhage was present. Smears and cultures were made to determine the bacterial flora in the conjunctival sac.

OPERATIVE PROCEDURE

The preoperative technic was the same as that followed in all surgical conditions on the eyeball in the Wilmer Institute. The night before the operation, the lashes are clipped, the skin of the lids and face is cleaned with liniment of soft soap, a drop of 10 per cent mild silver protein is instilled into the eye and washed out with boric acid, the eye is filled with 1:10,000 corrosive mercuric chloride ointment, and a sterile dressing is applied. The next morning the bandages are removed, and irrigations with mild silver protein and boric acid are given every hour until the time of the operation. When the patient is brought into the operating room his skin is again cleansed with liniment of soft soap, and the eye is irrigated. Sterile drapes are then used to cover the patient. The orbicularis oculi is paralyzed by infiltration of 1 per cent procaine hydrochloride, and the eye is anesthetized by the instillation of 0.5 per cent *p*-*n*-butylaminobenzoyl-dimethylamino-ethanol hydrochloride repeated four times at two minute intervals. The patient is then ready for the surgeon.

When one is operating from above, the eyeball is rotated downward and scissors are used to make a transverse incision in the bulbar conjunctiva, 7 mm. above the limbus and extending from the temporal to the nasal margins of the cornea. This flap is then turned back and dissected down to the limbus. A 000 catgut suture is placed at each corner of the flap, and each suture is carried through the opposite lip of the conjunctival incision, but these are not tied (fig. 1). The operator thus stands ready at all stages of the operation to close off the wound quickly if an emergency should require it. From this time onward, speed and accuracy are essential factors, for the eyeball will be opened widely and complications must be anticipated if not encountered. The tip of an Agnew spade knife is introduced at the upper limbus beneath the conjunctival flap, and carried into the anterior chamber for several millimeters (fig. 2). The knife is then plunged

downward through the iris tissue into the vitreous, so that the resultant incision is the full width of the knife, namely, 5 mm. (fig. 3).

The knife is then quickly withdrawn, and the closed de Wecker or Berens scissors are introduced through the corneal incision. The scissors are allowed to open slightly, so that one blade slides posteriorly to the iris, while the other blade lies on the anterior surface. A quick cut is made downward and inward from the temporal edge of the horizontal incision (fig. 4). The scissors are then carried over to the inner edge and another cut made down and out, so as to complete the triangle (fig. 5). The central portion of the iris tissue, which has been

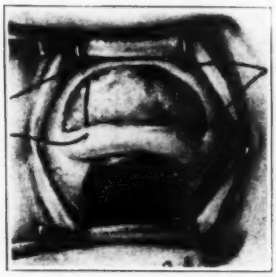

Figure 1

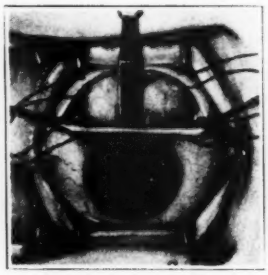

Figure 2

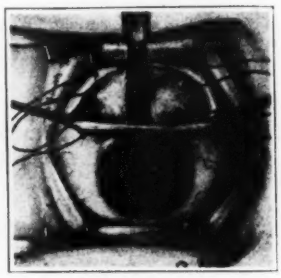

Figure 3

Fig. 1.—Dissection of conjunctival flap.

Fig. 2.—Introduction of Agnew knife.

Fig. 3.—Incision of iris.

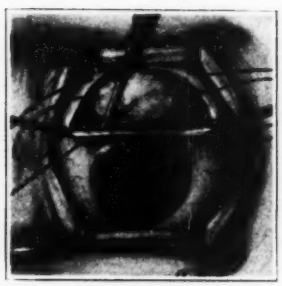

Figure 4

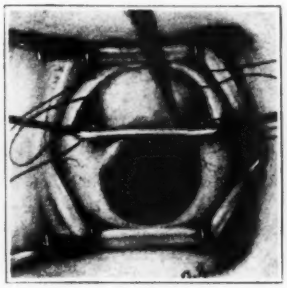

Figure 5

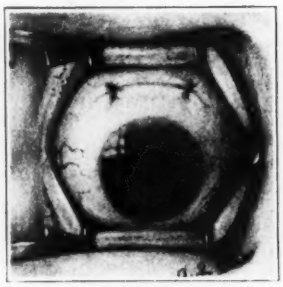

Figure 6

Fig. 4.—Initial cut with scissors.

Fig. 5.—Second scissors cut.

Fig. 6.—Result of operation.

excised, is removed with the scissors, and the sutures through the lips of the conjunctival wound are tied. During this entire time, the vitreous chamber has been widely opened, and when it is remembered that a previous inflammation of the globe has probably resulted in a very fluid vitreous, the absolute insistence on speed can be appreciated. Atropine, 1 per cent, is instilled into the eye, which is then filled with corrosive mercuric chloride ointment, and a pressure bandage is applied to prevent hemorrhage. The resultant pupillary opening usually measures about 5 mm. across the base of the triangle above, and 7 mm. from the center of

the base to the apex (fig. 6). The vertical distance can be increased as desired, even to the lower angle of the anterior chamber. The opening is thus wide enough to prevent it from closing unless filled by a considerable hemorrhage.

In some cases it is found advisable to place the base of the triangle below, owing to opacities of the cornea above. The procedure is then carried out in an exactly similar manner to that described, except that the incision into the anterior chamber is made at the lower limbus, at which point the Agnew knife is then introduced.

The chief factor in the postoperative care is the avoidance of hemorrhage. The patient is kept absolutely quiet in bed, and the dressing is not changed for forty-eight hours. At this time the eye is opened and inspected. If a small amount of hemorrhage is present, hot compresses can be applied to aid in its absorption. The eye is then dressed daily, care being taken to use considerable amounts of atropine in order to obtain its maximum effect on the iris.

SUMMARY

1. An operation for occluded pupil is offered which attempts to assure the maximum operative speed plus measures of safety against untoward effects.

2. A large pupillary opening results, unless considerable hemorrhage or iridocyclitis complicates the condition.

3. This procedure avoids marked traction on the ciliary processes, thus reducing to a minimum the danger of ensuing iridocyclitis or glaucoma.

4. No operation for an occluded pupil should be performed if the eye shows any sign of inflammation.

GLIOMA OF THE RETINA

REPORT OF A CASE WITH INTRACRANIAL EXTENSION

CARL WHEELER RAND, M.D.
LOS ANGELES

In a recent letter to the editor of *The Journal of the American Medical Association* Jean [1] discussed the inadequacy of intra-orbital operations in certain cases of glioma of the retina. He stated: "Glioma of the retina is a terrible disease and demands primarily brain and not eye surgery. When it kills, it does so practically always by direct extension of the tumor backward through the optic nerve to the brain. The surgical problem, then, is always to remove all the nerve possible."

That there is a close analogy between glioma of the retina and certain forms of glioma of the brain is apparent. In fact, the term glioma of the retina as used today is analogous to the term glioma of the brain as used a decade or more ago. Since the advent of newer staining methods, gliomas of the brain have been much more definitely classified, the classification depending on the type of cell predominating. Likewise retinal gliomas are beginning to be subdivided into various groups, depending largely on the type of cell predominating as shown by newer methods of silver impregnation.

The older terminology, glioma exophytum or tuberosum, glioma diffusum or planum and glioma endophytum, is gradually going out of use. Flexner [2] (1891) was the first to describe one of these types of tumor as a neuro-epithelioma. Later (1922) Verhoeff [3] used the term retinoblastoma for another type. In 1926 these newer terms were adopted by the American Ophthalmological Society as most suitable to describe the condition. Hu [4] presented the reports of 2 cases of neuro-epithelioma of the retina with metastases and discussed at some length the pathologic terminology of these so-called retinal gliomas. He considered that while termed gliomas they are different from ordinary glioma of the brain in their rapidity of growth, their greater tendency toward metastasis and their lack of glial fibers which may be demonstrated by ordinary staining methods. He mentioned the "rosette"

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1. Jean, G. W.: Glioma of the Retina, J. A. M. A. **100**:1793 (June 6) 1933.
2. Flexner, S.: A Peculiar Glioma (Neuro-Epithelioma?) of the Retina, Bull. Johns Hopkins Hosp. **2**:115, 1891.
3. Verhoeff, F. H.: Primary Intraneural Tumors (Gliomas) of the Optic Nerve, Arch. Ophth. **51**:120 (March) 1922.
4. Hu, C. H.: Neuro-Epithelioma (Glioma) of Retina with Metastases, Am. J. Path. **6**:27 (Jan.) 1930.

arrangement of certain cells as described by Flexner [2] and drew an analogy to the similar arrangement of certain primitive spongioblasts as emphasized by Bailey and Cushing.[5] He called attention to the fact that such terms as neuro-epithelioma or glioma of the retina are inadequate to designate the type of tumor in question:

> Since the retinal epithelium is formed by the budding off of the medullary epithelium which later develops into the central nervous system, we may expect to find tumors of the brain and the spinal cord which are analogous to those found in the retina. This is indeed the case. In the so-called neuro-epithelioma of the brain, structures similar to the rosettes of the retinal tumors are found. In the tumors called medulloblastomas by Bailey and Cushing both neuroglia cells and neuroblasts may be identified. These tumors, furthermore, present the same embryonal characteristics of the retinal tumor in their rapid growth, lack of cell differentiation, inconsistent presence of one or another element and tendency toward spreading into the meninges.

Grinker [6] recently reassembled the pathologic evidence in retinal tumors and classified the gliomas under three main headings:

1. Medullo-epitheliomas, which arise from the medullary epithelium, the cells of the primitive neural tube and the primitive retinal epithelium. These tumors are mixtures of cell forms, being composed primarily of medullary epithelium but containing retinoblastomatic and spongioblastic cells and exhibiting neuro-epithelial rosettes. These tumors are said to be exceedingly rare.

2. Retinoblastomas (retinomas and retinocytomas), which are composed of retinoblastic cells. These cells are supposed to be bipotential and capable of developing "along the neuroblastic series to form ganglion cells, or along the spongioblastic series to form adult glia." Neuroblasts have never been found in this type of retinal tumor. The neuroblastomas compose the vast majority of retinal gliomas. They usually arise from the posterior part of the retina, breaking through early and showing an extensive power of infiltration. These tumors extend into the orbit, invade the base of the brain and show distant metastases. They are the only known neoplasms of glial origin which metastasize to different parts of body. In some ways they resemble the medulloblastomas of the brain which, to a lesser degree, spread along the meninges of the brain and spinal cord presumably by a process of implantation. This power of metastatic growth in retinal blastomas has not been adequately explained although the embryonic characteristics of

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5. Bailey, P., and Cushing, H.: A Classification of the Tumors of the Glioma Group on a Histogenetic Basis with a Correlated Study of Prognosis, Philadelphia, J. B. Lippincott Company, 1926.

6. Grinker, Roy R.: Tumors of the Retina, in Penfield, W.: Cytology and Cellular Pathology of the Nervous System, New York, Paul B Hoeber, Inc., 1932, vol. 3, p. 1043.

the cells would indicate a high degree of malignancy. Urra,[7] in discussing retinoblastomas, described three zones of cellular development: (*a*) an external zone composed chiefly of retinoblasts, (*b*) a middle zone composed chiefly of spongioblasts and (*c*) an internal zone composed chiefly of adult astrocytes.

3. Neuro-epitheliomas, first described by Flexner [2] and Wintersteiner.[8] For the most part they are composed of primitive spongioblasts arranged in true rosettes. Identical tumors in the brain have been described, but are very rare. Bucy and Muncie [9] described an example in the cerebellum which they stated was the fifth authentic case on record. These retinal neuro-epitheliomas are less malignant than retinoblastomas. They are said to arise from the external nuclear layer, or in the pure form from the ciliary process. Bailey and Cushing [5] stated that these tumors might correctly be called "spongioblastoma primitivum retinae."

That these retinal tumors kill by direct extension along the optic nerve to the brain or by metastasis, or both, has long been known. Reese [10] summed up the results of microscopic examination of 119 eyes enucleated for glioma of the retina with no apparent extra-ocular extension. In 63 (52 per cent) the optic nerve had already been invaded by gliomatous tissue. Of these, 51 (81 per cent) showed that the optic nerve had been severed distal to the extension of the tumor. In other words, gliomatous tissue still existed at the cut end of the optic nerve in 43 per cent of the entire series of 119 cases. The patients were naturally doomed unless they were again operated on and the nerve removed intracranially beyond the point of invasion. Reese commented on the increasingly good results reported for enucleation in cases of glioma of the retina. In 1868, Hirschberg [11] reported cure in 5 per cent of a series of 75 cases. In 1897, Wintersteiner [8] reported cure persisting two years after enucleation in 13 per cent of a series of 497 cases. In 1911, Adam [12] reported cure in 57 per cent of a series of 46

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7. Urra, F.: Ueber die feine Gewebstructuren des Glioma der Netzhaut, Arch. f. Ophth. **112**:133, 1923.

8. Wintersteiner, H.: Das Neuroepithelioma Retinae, Vienna, Franz Deuticke, 1897.

9. Bucy, P., and Muncie, W.: Neuroepithelioma of the Cerebellum, Am. J. Path. **5**:157 (March) 1929.

10. Reese, A. B.: Extension of Glioma (Retinoblastoma) into the Optic Nerve, Arch. Ophth. **5**:269 (Feb.) 1931.

11. Hirschberg, J.: Anatomisch Untersuchungen ueber Glioma Retinae, Arch. f. Ophth. **14**:30, 1868.

12. Adam, C.: Statistisches, klinisches und anatomisches ueber das Glioma retinae, Ztschr. f. Augenh. **25**:330, 1911.

cases, while in 1916, Leber,[13] reporting a series of 28 cases, likewise showed cure in 57 per cent. If it is true that 43 per cent of gliomas of the retina have extended intracranially at the time of enucleation, 57 per cent would seem to represent the maximum number of cures which have been obtained by simple enucleation or exenteration of the orbit. These figures could doubtless be improved if enucleation were done earlier in the disease.

It might be well to recall that a group of closely allied tumors of the optic nerve have been described. These tumors may be chiasmal in origin or may arise from the optic nerve proper and extend back to the chiasm. The first one reported was described by Heymann [14] as a neuroma nervi optici extending to the chiasm. The first attempt at intra-orbital enucleation for "sarcoma" of the nerve was made by Ritterich [15] and resulted fatally. An enormous enlargement of the chiasm was found at autopsy.

These tumors differ pathologically from the various types of gliomas of the retina under discussion. They often arise from the sheath of the nerve and may be found when general neurofibromatosis exists elsewhere. I have seen a case of primary tumor of the chiasm associated with Recklinghausen's disease. Other tumors, such as endotheliomas, or meningiomas as they have more recently been called, are similar and may occur both intra-orbitally and intracranially along the course of the optic nerve.

There is some evidence that such tumors may progress in either direction through the optic foramen; a primary intra-orbital growth may secondarily become intracranial, or a primary intracranial growth may secondarily become intra-orbital. Their progress is much slower than the true retinoblastomas under discussion. Byers [16] reported a case of endothelioma in which the patient was operated on for an intra-orbital tumor and died ten years later from intracranial extension of the growth. Pagenstecher [17] reported a similar case of Steffan's [17a] in which the patient lived twenty-five years after the removal of the orbital tumor.

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13. Leber, T.: Die Geschwülstbildungen der Netzhaut, in Graefe-Saemisch: Handbuch der gesamten Augenheilkunde, ed. 2, Leipzig, Wilhelm Engelmann, 1916, vol. 7, chap. 15.

14. Heymann, A.: De neuromate optici, Berlin, 1842.

15. Ritterich, F. P.: Weitere Beiträge zur Vervollkommnung der Augenheilkunst, Leipzig, C. F. Winter, 1861, p. 57.

16. Byers, W. G. M.: Primary Intradural Tumours of the Optic Nerve, Stud. Roy. Victoria Hosp., Montreal **1**:3, 1901.

17. Pagenstecher, A. H.: Ueber Opticus Tumoren, Arch. f. Ophth. **54**:300, 1902.

17a. Steffan, P.: Steffan'sche Augen-Heilanstalt in Frankfurt am Main, Jahresberichte **12**:33, 1874.

Bennett [18] reported an unusual surgical procedure for intradural tumor of the optic nerve. In his patient, a boy aged 11 years, a tumor of the left optic nerve developed at the apex of the orbit, causing total loss of vision in the left eye. At the time of enucleation 1.3 cm. of apparently normal nerve was attached to the eyeball. The tumor, however, lay posterior to this, for it could be felt by the finger extending down into the optic foramen. "In order to remove all of this mass a small gouge was inserted into the apex of the orbit, and the whole of the tumor was removed with 1 cm. of healthy looking nerve at the posterior end; in fact, the nerve was partially torn away at or very near the chiasm." The child recovered, although for a time he had temporal hemianopia of the right eye, "due to the dragging on the nerve at the time of operation." Bennett did not state what degree of vision remained as a result of this unusual evulsion.

Approaches to the optic chiasm are now frequent procedures with neurologic surgeons. These approaches are, in the great majority of cases, carried out to expose and deal with the chiasmal lesions caused by various types of pituitary tumors. Primary tumors of the intracranial portion of the optic nerve have been operated on by Dandy,[19] Martin and Cushing [20] and others; these measures, however, were not used for primary retinal gliomas with intracranial extension. Dandy [19] stressed the fact that a high proportion of intra-orbital tumors of the optic nerve may and do extend into the cranial chamber. He expressed the belief that local operations on the orbital part of such tumors not only are futile, but in addition give the patient a false sense of security until it is too late to circumvent the growth by an intracranial approach. He stated that it should be determined early whether or not there is intracranial extension or an intra-orbital tumor. If any exists "only an intracranial operation which aims at the removal of the tumor offers the patient any chance of preservation of life or vision."

Dott and Meighan [21] were probably the first to resect the optic nerve intracranially for extension of glioma of the retina. They reported before the Ophthalmological Society of the United Kingdom at Edinburgh that they had successfully resected the optic nerve at the chiasm in a case of glioma of the retina with intracranial extension along this structure. Apparently their incentive to operate was the fact that on

18. Bennett, H. P.: Case of Primary Intradural Tumor of the Optic Nerve, Brit. M. J. **1**:1041, 1905.

19. Dandy, W. E.: Prechiasmal Intracranial Tumors of the Optic Nerves, Am. J. Ophth. **5**:169 (March) 1922.

20. Martin, P., and Cushing, H.: Primary Gliomas of the Chiasm and Optic Nerves in Their Intracranial Portion, Arch. Ophth. **52**:209 (May) 1923.

21. Dott, N. M., and Meighan, S.: Intracranial Resection of the Optic Nerve in Glioma Retinae, Am. J. Ophth. **16**:59 (Jan.) 1933.

examining the cut end of the nerve after enucleation, which had been performed fourteen days before, infiltration by tumor was found at the point of section. Examination of the cut end of the nerve at the chiasm did not reveal any invasion by tumor, and it is presumable that they succeeded in circumventing the growth. The child recovered and seven months later was reported as being in excellent health.

Occasional cures or regressive changes in gliomas of the retina have followed high voltage roentgen therapy. Stewart [22] summed up the results as follows:

> In cases of neuro-epithelioma of the retina and retinoblastoma considerable regressions have followed irradiation. Recurrence is the rule. Some patients with retinal gliomas have been cured by external irradiation. The tumors were small. Irradiation seems most successful when after one of these usually bilateral gliomas has attained such a size that it becomes clinically discoverable attention is directed to the opposite eye, in which a small tumor is detected. Irradiation of these smaller tumors may result in cure with conservation of vision. Successes have been reported by Axenfeld, di Marzio and Salvatori, Moore and Knopp and Ludin. Benedict reported regression of retinoblastomas. Very large doses were required. A posterior cortical cataract occurred as a later complication.

REPORT OF A CASE

Glioma of the retina in a girl aged 4 years and 7 months. Enucleation of the eye performed four months previously with rapid local recurrence. Intracranial resection of the optic nerve showed disease had already invaded the chiasm and base of the brain. Death.

History.—The patient was first seen at the Children's Hospital on June 17, 1933. She was an only child. The father and mother were both living and well, and the past history was irrelevant.

When she was about 2 years of age her mother first noticed a white spot in the pupil of the left eye. Gradually the pupil became white. Various physicians said that the eye was blind. When the child was 3 years and 9 months of age the left eyeball began to enlarge. The pupil on this side also began to enlarge. She was seen by Dr. Simon Jesberg, who, on Nov. 27, 1932, enucleated the left eyeball for glioma of the retina (fig. 1). At that time the optic nerve was resected back as far as possible, but microscopic evidence of tumor at the cut end of the nerve was present. It was evident that the tumor extended intracranially. She was then given two courses of high voltage roentgen therapy by Dr. Albert Soiland. The first course extended from December 2 to 20, and the second course from May 11 to 26, 1933; 185 kilovolts, a focal skin distance of 50 cm. and 0.5 mm. copper and 1 mm. aluminum filters were used. The patient received a total dosage of 1,600 roentgens.

Examination.—At the time of admission to the Children's Hospital there was an advanced recurrence of the tumor. The new growth occupied the entire socket. The tumor was quite firm and round, pushing the eyelids forward and giving the appearance of exophthalmos. The outer surface of the tumor was covered with

22. Stewart, F. W.: Radiosensitivity of Tumors, Arch. Surg. **27**:1061 (Dec.) 1933.

normal conjunctiva. The veins of the upper lid were dilated. Vision of the right eye was impaired, the acuity being 20/50+. Perimetry showed moderate constriction along the temporal field of this eye, suggesting that chiasmal involvement had already taken place (fig. 2). The extra-ocular movements on the right were normal. The right fundus showed the disk plainly; the margins were sharply outlined and the optic cup distinct. The disk was a little pale, although hardly enough to suggest primary atrophy. A general neurologic examination gave entirely negative results.

Roentgenographic studies were made in special positions to compare the size of the optic foramina. The optic foramen reaches its maximum size at about 3 years of age, and at this time the child was 4 years and 7 months of age. The

Fig. 1.—Gross specimen of the enucleated (left) eyeball. An extensive growth of tumor, filling about one third of the vitreous space, is seen. The arrow indicates the point at which the tumor has broken into the anterior chamber. The nerve head is enlarged, showing infiltration of the tumor into this structure. Enucleation was performed approximately two years after the first symptoms appeared. The cut end of the optic nerve showed invasion of the tumor process both macroscopically and microscopically; × 10.

right optic foramen measured 4.5 mm. in A diameter and 5 mm. in B diameter; the left optic foramen, 4.5 mm. in A diameter and 5.5 mm. in B diameter. Thus the left foramen was a little larger than the right in B diameter, but there was not enough variation to be of diagnostic value. There was no undue erosion.

Examination of the blood and urine and a general physical examination gave negative results.

Operation.—On July 5, 1933, the patient was operated on by an intracranial approach in the hope of severing the optic nerve at the chiasm beyond the extension

of the growth. A frontal flap was turned on the left side, as is the custom in a pituitary approach. The roof of the left orbit was exposed and removed. The orbit contained a tightly packed tumor, which was quite adherent to the walls. It was impossible to remove this tumor in one mass. The capsule was opened, and the contents were scooped out. These consisted of grayish, soft, vascular tissue. Piece by piece the capsule was then picked up and dissected out. In doing this the conjunctiva covering the tumor anteriorly was unfortunately torn so that a portal of infection was provided. The optic chiasm was uncovered by opening the dura. The left optic nerve was seen to be considerably larger than the right up to a point about 0.5 cm. from the chiasm. Here it tapered down and appeared grossly normal. The nerve was picked up with a blunt hook and cut across at the chiasm with scissors. The roof of the optic foramen was then removed, and the remaining diseased gliomatous stump of optic nerve was taken out. The wound was closed, drainage being provided through the socket. The entire procedure was slow and difficult.

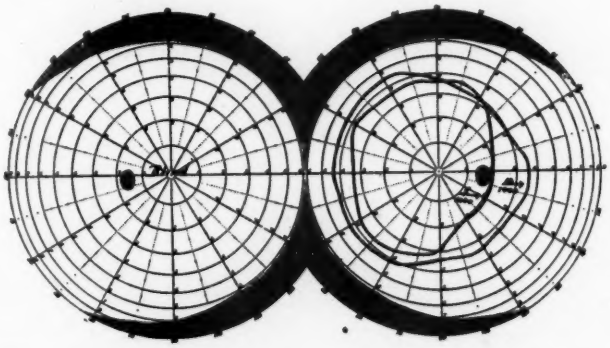

Fig. 2.—Visual field showing general contraction on the right. The contraction is greater on the temporal side. Vision of the right eye was 20/50+. This contraction was due to invasion of the right optic nerve by the tumor. The left eye had been enucleated.

Following operation a cerebrospinal fluid leak developed. At times the flow of fluid was copious, at other times scant. Ultimately the fluid became infected, giving rise to a basal meningitis. The patient died on August 3.

Dr. C. M. Hyland, pathologist for the Children's Hospital, made the following report on the microscopic examination of tumor tissue (July 18, 1933) and on the observations at autopsy:

Examination of Tumor Tissue.—Tissue from the tumor mass obtained at operation was composed entirely of the same type of cell with scant stroma and showing necrosis in many areas (fig. 3). The cytoplasm content was small and the nuclei showed variations in staining reactions. An occasional mitotic figure was observed. No classic formation of pseudorosettes was observed; however, in places clumps of cells were found which suggested this possibility. Sections taken from the peripheral end of the optic nerve showed infiltration by the tumor process.

Sections taken from the central end of the optic nerve showed marked infiltration of tumor tissue which extended completely around the nerve and was found

Fig. 3.—Section of a recurrent tumor removed from the orbit six months after enucleation. This reveals a very cellular growth with areas of necrosis and some tendency to rosette formation of tumor cells in certain fields. Hematoxylin and eosin; × 85.

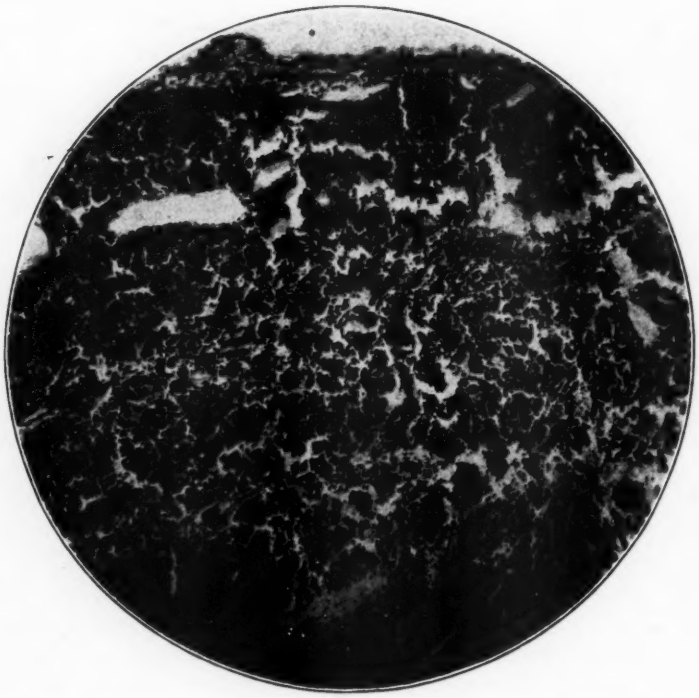

Fig. 4.—Section of the cut end of the optic nerve at the chiasm, showing almost complete infiltration of the stump by tumor cells. Hematoxylin and eosin; × 70.

beneath its sheath (fig. 4). And at one point, it extended deeply between the bundles of nerve fibers. The cells comprising the nerve infiltration resembled those found in the tumor sections.

Autopsy.—Brain: The weight was 1,150 Gm. As the calvarium was removed, considerable hyperemia and edema of the leptomeninges were noted. The pia was opaque; this was particularly evident along the course of the vessels. A small amount of exudate was visible beneath the pia-arachnoid, and there was an adhesion between the pia-arachnoid and the superior portion of the orbit. As the brain was removed, it was noted that there was a marked pressure furrow at the foramen magnum, with much edema of the brain tissues at this point. The left optic nerve was missing (removed at operation); the end of this nerve did not extend beyond the chiasm more than 1 mm.

Fig. 5.—Section of the optic chiasm cut tangentially. Marked infiltration of the left optic nerve, extending well into the chiasm, is seen. There is tumor invasion beneath the sheath of the right optic nerve as well as posteriorly in the chiasm. The optic tract also shows tumor invasion. Hematoxylin and eosin; × 10.

Optic Tracts: The optic tracts removed at autopsy consisted of the entire intracranial portion and a few millimeters of the interosseous portion of the right nerve. The greater share of the intracranial portion of the left nerve had been removed (operation), and not more than 1 mm. of this nerve was present distal to the optic chiasm.

The optic chiasm was present in its entirety, and the optic tracts were removed at their entrance into the thalamencephalon and the mesencephalon. Under low power magnification one could trace the right nerve throughout its entire extent, but the left nerve showed a loss of continuity in its course from the sectioned portion to its entrance into the brain (fig. 5). Examination with higher magnification showed a remnant of the intracranial portion of the left optic nerve, but most of this structure had been destroyed by a diffuse infiltration of tumor tissue; at the level of the section examined there was complete loss of continuity in the nerve tract.

The tumor tissue was very vascular, showing well formed blood vessels in its substance. It was composed of rather small cells containing nuclei which took a dense stain and little cytoplasm. The stroma was scant, and necrosis was present in some areas of the tumor. The cells described varied somewhat in size and shape, but tended to be round or slightly ovoid. Groups of these cells also existed posterior to the chiasm and beneath the arachnoid covering of the right optic nerve.

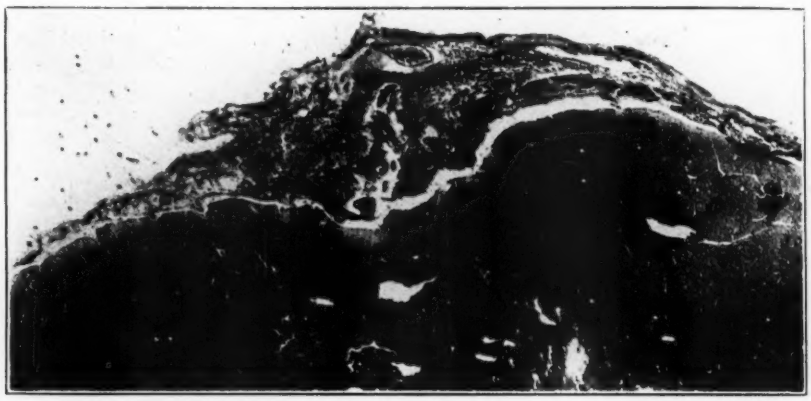

Fig. 6.—Section from the base of the temporal lobe, showing invasion of the subarachnoid space by tumor cells. Hematoxylin and eosin; × 10.

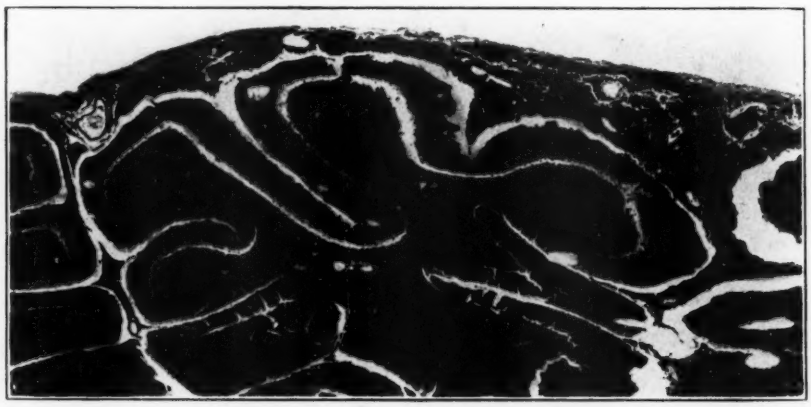

Fig. 7.—Section from the floor of the cerebellum, showing invasion of the subarachnoid space by the tumor. Hematoxylin and eosin; × 10.

In places, they extended rather deeply into the nerve tissue and tended to follow the walls of blood vessels into the deeper portions. In an occasional small vessel tumor emboli were seen. Sections taken from the inferior surface of the cerebrum just posterior to the optic chiasm showed, beneath the arachnoid, masses of cells identical with those just described. These cells had greatly distended the spaces between the membrane and the brain. Occasionally they extended slightly downward or were seen within the brain substance; however, for the most part, the growth was on the surface of the brain (fig. 6). Sections taken from the cere-

bellum showed at one point the same type of tumor tissue found on the inferior surface of the cerebrum and about the optic tracts. At one point a mass of cells extended rather deeply into the cerebellar tissue (fig. 7).

The diagnosis of the condition found in the optic tracts was: gliomatous infiltration of both optic nerves and of the arachnoid on the inferior surface of the cerebrum and cerebellum.

COMMENT

How radical should the treatment be in cases of glioma of the retina? Naturally, much depends on the stage at which the lesion is recognized. If treatment is attempted in an early stage it is probable that simple enucleation will suffice to cure the large majority of patients. Unfortunately, enucleations are seldom done before the growth has become extensive enough to jeopardize or cause loss of vision. Few parents are willing to have what seems to them to be a normal eye removed. When blindness or exophthalmos has developed, their reaction to enucleation is quite different. It would seem that on the average only 57 per cent of patients with glioma of the retina are ultimately cured by enucleation or exenteration of the orbital contents. Occasionally cures have been reported in rather advanced cases following enucleation and radium therapy; these are rare. It is the fate of the remaining 43 per cent which is of interest.

In the case presented here the disease had already been present more than two and a half years before any operative attempt was made. Enucleation was not carried out until the tumor had largely filled the eyeball and broken through the iris. Exophthalmos at that time was marked and blindness complete. On removal of the eye study of the cut section of the optic nerve showed invasion of this structure by tumor. Even then no attempt was made to remove the nerve further until another six months had elapsed. By then the tumor had refilled the orbital cavity and also had extended well along the optic nerve. To be sure at operation the nerve at the chiasm appeared normal, although at the optic foramen it appeared enlarged. Microscopic section, however, showed that tumor tissue had already invaded the chiasm. A rather poor perimetric chart made shortly before this intracranial operation tended to bear this out, as there was considerable constriction of the temporal field in the right eye. A fatal outcome was inevitable. There is reason to believe that this might have been prevented had the child been operated on earlier.

Careful judgment is required in considering what the criterion should be for advocating intracranial section of the optic nerve. If ophthalmologists could establish from examination and symptoms that the tumor had already penetrated the optic foramen, primary intracranial section of the nerve would be indicated. Roentgenographic studies of the optic foramina should be made in every case, and enlargement of

the suspected foramen would seem sufficient evidence to justify the assumption that the tumor had already invaded this structure. Here again, primary intracranial section and removal of the optic nerve would seem indicated. On the other hand, if the ophthalmologist believes the tumor has not extended beyond the orbital contents, enucleation might reasonably be expected to cure the condition. Section of the cut nerve should be studied immediately to determine whether or not all tumor tissue has been removed. If all has been removed, further operation is not indicated. In case invasion of the stump by tumor tissue is found to exist, intracranial section of the optic nerve at the chiasm and the entire removal of the stump are in order. In the present case the dura was not closed carefully enough, and a cerebrospinal fluid leak occurred with resulting secondary infection. If enucleation is carried out first, a few days should probably be allowed to elapse until healing has sealed off the stump. Should the intracranial section of the nerve be carried out first, secondary enucleation could probably be accomplished at the same operation. Before this is done, however, tight closure at the apex of the orbit should be carried out to prevent the occurrence of a cerebrospinal fluid fistula.

It is not at all probable that a large proportion of patients presenting intracranial extension along the optic nerve can be saved even by the radical surgical measures outlined. It is, however, entirely probable that some of them can be cured, thus justifying the procedure.

CONCLUSIONS

The earliest possible enucleation is advocated in all cases of unilateral glioma of the retina.

Careful study of the sectioned nerve should be made at the time of enucleation.

In certain cases in which the tumor has extended along the optic nerve intracranially, resection of this structure from the chiasm outward is indicated.

From my observations in a fatal case intracranial extension of glioma of the retina occurs in two stages: (*a*) along the optic nerve, finally breaking through the sheath, and (*b*) extending intracranially along the base of the brain in the subarachnoid spaces.

PHTHIRIASIS PALPEBRARUM

REPORT OF A CASE

REUBEN FRIEDMAN, M.D.

AND

CARROLL S. WRIGHT, M.D.

PHILADELPHIA

De Schweinitz defined phthiriasis palpebrarum (phthiriasis ciliorum, blepharitis pediculosa, pediculosis ciliorum) as that condition which "occurs when the pediculus pubis or crab louse forsakes its seat of predilection and finds a habitat among the eyelashes."

According to Ball,[1] this condition "is uncommon in this country." Schwenk,[2] however, reported having personally observed 20 cases among 20,319 patients at the dispensaries of the Wills and Pennsylvania Hospitals in Philadelphia, during ten years' clinical service, the ratio being 1:1,016 patients. He further stated: "I think the infrequency of these cases is greatly overrated. Hirschberg reports having seen only three cases in 40,000 patients. Despagnet reports only two cases in twelve years in the large clinic of Galezowski. There are two cases out of 10,000 cases reported by Dr. de Schweinitz as having been seen in Dr. Norris's clinic at the Hospital of the University of Pennsylvania. Julien . . . says that it occurs but once in 5,000 cases of eye diseases. In contradiction to this, Dr. Charles W. Allen reports eighty-five cases of pediculi ciliaris out of 5,974 patients seen at the Good Samaritan Dispensary, New York City, during 1891."

Oddly enough, a refutation of Schwenk's belief that "the infrequency of these cases is greatly overrated" is found on page 123 of the volume [2] containing his report. It is stated there that a total of 12,614 patients were treated in the dispensary and wards of the Wills Eye Hospital in 1894. Of this number, 898 are listed as suffering from diseases of the eyelids and eyebrows; yet only 1 specific instance of "phtheiriasis ciliaris" is reported in this group. It would seem, therefore, that, at least for 1894, the ratio of occurrence of this disease at the Wills Eye Hospital was 1:12,614 patients.

From the Department of Dermatology and Syphilology, Temple University School of Medicine and Hospital, service of Dr. Carroll S. Wright.

1. Ball, J. M., in Wood, C. A.: American Encyclopedia of Ophthalmology, Chicago, Cleveland Press, 1912, vol. 2, p. 1,031.

2. Schwenk, P. N. K.: Pediculi Ciliaris: with a Report of a Number of Cases of "Crab-Lice" in the Eyelashes, Wills Eye Hosp. Rep. **1**:69, 1895.

Fig. 1.—Phthiriasis palpebrarum, with ova on both the upper and the lower eyelashes; ova may also be seen on the hairs near the inner border of the eyebrow.

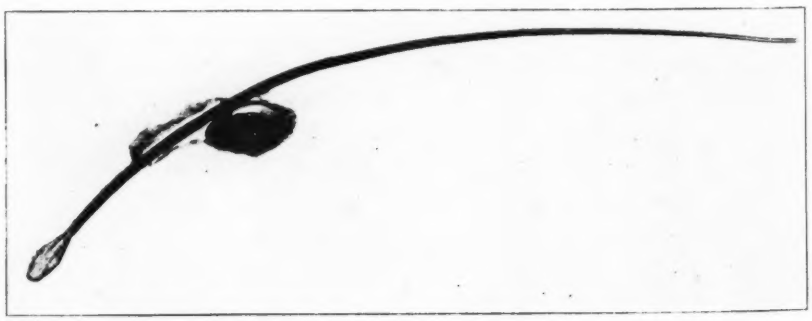

Fig. 2.—Ovum of pediculus pubis attached to a lash of the lower eyelid; the ovum contains an embryo; the operculum is intact; reduced from a magnification of × 20.

More recent writers—de Schweinitz,[3] Fuchs [4] and May,[5] for example—as well as most other authorities, agree that the disease "is rare and is found almost exclusively in children." On the other hand, Professor Aubaret [6] of Marseilles, in an exhaustive article on blepharitis, stated that "pediculosis of the lids is today more frequent than one would expect, in town and in country, in crowded slums and asylums and among neglected, dirty children."

The following case of phthiriasis palpebrarum is reported for two reasons: first, because of its occurrence in a 19 year old college student who, incidentally, came to the clinic not because of this condition but because of acne, and second, because we were able to obtain a good photograph of the affected eyelids (fig. 1). Photographic illustrations of this disease are rare.

REPORT OF CASE

O. S., a white 19 year old male college student, was first seen on Jan. 5, 1934, at Dr. Wright's clinic in the Temple University Hospital, where he came to receive treatment for acne. During the course of the examination of the face, blepharitis was noted, affecting only the right eye. Closer inspection of the margins of the eyelids and the eyelashes aroused the suspicion that the peculiar dotlike bodies observed thereon were ova, a suspicion that was strengthened by the use of a magnifying glass and confirmed by microscopic examination of an extracted eyelash to which an ovum was attached (fig. 2). As many as four ova were found attached to some lashes of the upper eyelid. No pediculi were found.

The patient was requested to remove all his clothing, and examination revealed pediculosis pubis of moderate severity. On being questioned, he stated that he was right handed, which may explain why only the right eyelids and eyebrow were involved.

Beside the presence of Pediculus pubis and ova on the pubic hairs and the presence of ova on the upper and lower right eyelashes and right eyebrow, ova were also found on hairs about the umbilicus, on the sternum, in both axillae and on both thighs as far down as the knees. A living female pediculus was recovered from a hair immediately above the right knee.

The blepharitis cleared up within ten days under treatment with ointment containing 1 per cent yellow mercuric oxide.

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3. de Schweinitz, G. E.: Diseases of the Eye, ed. 10, Philadelphia, W. B. Saunders Company, 1924, p. 182.

4. Fuchs, E.: Text-Book of Ophthalmology, translated by Alexander Duane, ed. 7, Philadelphia, J. B. Lippincott Company, 1923, p. 413.

5. May, C. H.: Manual of the Diseases of the Eye, ed. 13, New York, William Wood & Company, 1930, p. 47.

6. Aubaret, E.: Blepharitis: Etiology and Treatment, Arch. Ophth. **3**:762 (June) 1930.

ANATOMIC AND CLINICAL MANIFESTATIONS OF NECROSIS IN EIGHTY-FOUR CASES OF CHOROIDAL SARCOMA

BERNARD SAMUELS, M.D.
NEW YORK

There are sarcomatous globes in which instead of an attack of ordinary glaucoma there is a sudden outbreak of extremely severe iridocyclitis which may lead to atrophy of the organ. Anatomic examination in such instances reveals that the tumor has become necrotic. The necrosis is due to deficient nutrition, as the new blood vessels are large and there are no capillaries. The cells, which gain their nourishment by osmosis, perish when they are too far away from the blood supply. As a result of the death of the cells cytotoxins are developed which in themselves have a necrotizing influence on the tissues of the globe. The ciliary body, the iris and the cornea above all are affected.

In order to understand the important rôle that necrosis and toxins may play in the pathologic and clinical pictures of a sarcoma of the choroid one should bear in mind the four anatomic stages into which its life is divided. In the first stage the tumor is oval or flat and is confined to the choroid, being compressed between the sclera and the lamina vitrea. This stage may last many months and cause no symptom other than disturbance of vision. The tumor acts as a foreign body in the stroma of the choroid and interferes with the flow of blood toward the vortex veins. When it is located in the posterior part of the eye, the engorgement of the choroidal vessels is apt to be more marked on the side of the tumor nearer the optic nerve. The engorged vessels are sometimes seen clinically as a narrow reddish zone. During the first stage transudation of serum may take place from the engorged vessels of the choroid and cause detachment of the retina. The second stage is ushered in by disappearance of the lamina vitrea at one point, forming an opening through which the tumor proliferates in fluid that has collected under the retina. The proliferation assumes a nodular shape in the subretinal fluid because the pressure is equal in all directions. The flat part of the tumor is spoken of as the base, the constriction at the lamina vitrea as the neck and the nodule as the head. The entire formation which is typical of choroidal sarcoma is familiarly known as a mushroom-shaped sarcoma. Occasionally a low, diffuse sarcoma that replaces an extensive area of the uveal tract or a large hemispherical sarcoma is encountered over which the lamina vitrea has been elevated

Read before the Oxford Ophthalmological Congress, Oxford, England, July 8, 1933.

but has not disappeared. The third stage consists in the propagation of the tumor through the sclera and the development of an extra-ocular nodule in the loose tissues of the orbit. The fourth stage is that of metastasis to distant parts of the body. However, metastasis may occur at any stage, even when the tumor is microscopic.

From the standpoint of effect on the tension of the globe, the life of a sarcoma is divided into two stages. In the first stage the tension remains normal, whereas in the second there is a rise of tension. The preglaucomatous stage corresponds to the first anatomic stage, in which the tumor is flat and the retina is in situ or is very slightly detached. There is no cause for increase in tension in this stage because as the tumor grows the vitreous body disappears by pressure atrophy. The glaucomatous stage corresponds to a late period in the second anatomic stage, by which time the retina has become extensively detached. The subretinal fluid transudes from the choroid under a pressure higher than that in the vitreous body, so that not only is the vitreous made to atrophy but what is left of it is driven forward, pushing ahead the lens and the iris at the expense of the aqueous chamber.

The pressure caused by increase in intra-ocular tension has a deleterious effect on the various structures of the eye. It produces recession of the cribriform fascia, and with this atrophy of the nerve fibers. It impedes the circulation of the blood and lymph, among the results of which is atrophy of the iris and ciliary processes. During the glaucomatous stage the tumor may become necrotic, so that the toxins act on tissues that may have suffered already from glaucoma.

That many patients first consult the surgeon after the tumor is in a state of necrosis or do not submit to enucleation of the globe until necrosis has occurred is borne out by the fact that of one hundred and six sarcomatous eyes that were examined microscopically in the Eno Laboratory at the New York Eye and Ear Infirmary from April 27, 1927, to May 16, 1933, there were thirty-one that were highly necrotic. Of these, there were twenty in which the following diagnoses indicate that the presence of a necrotic sarcoma was not suspected:

	Cases
Absolute glaucoma	11
Hemorrhagic glaucoma	1
Acute glaucoma	1
Secondary glaucoma	1
Glaucoma after detachment of the retina	1
Iridocyclitis	1
Traumatic cataract	1
Staphyloma of the sclera	1
Keratitis bullosa, acute iritis, complicated cataract, secondary glaucoma	1
Atrophic globe	1
Total	20

A correct clinical diagnosis of choroidal sarcoma in a state of necrosis is as important as the diagnosis of the tumor in any other state. The ordinary textbook on ophthalmology does little more than mention that intra-ocular sarcomas may undergo necrosis. Since the observations that were made by Fuchs [1] in a paper entitled "Concerning Sarcoma of the Choroid, with Remarks on Necrosis of the Uvea," not much has appeared in the literature relating to the destructive power of toxins thrown off from necrotic sarcomas and the symptoms that they cause. I discussed the topic incidentally in a paper entitled "Necrosis of the Iris." [2]

The main purposes of the work reported in this paper were, first, to make a microscopic study of the damage that may be done to the different structures of the globe by the irritant action of toxins generated by necrotic sarcomas and, second, to correlate the anatomic findings with the clinical manifestations.

MICROSCOPIC STUDY

The material on which the anatomic study was based consisted of sections from eighty-four globes every one of which revealed a necrotic sarcoma. Of some eyes there were many sections, whereas of others there were few, as would be expected in so large a series gathered from many parts of the world. However, the general conclusions should be accurate enough to be of scientific value.

In order to avoid confusion, there follows an explanation of what is meant by necrosis:

1. Necrosis of a sarcoma. Almost any small sarcoma of the uvea shows minute areas that stain poorly as an expression of the death of a group of cells. Toxins from such small areas apparently cause no symptoms. The tumors here considered were mostly large ones in which whole lobules were completely decayed, generally of sufficient size to be recognized in the gross preparations by their dirty, indefinite color. In a number of sarcomas the necrotic areas were surrounded by a coal-black pigmented border that stood out prominently against the enclosed, poorly staining necrotic area.

2. Necrosis of the tissues. Necrosis of structures of the eye refers, unless otherwise stated, to death and disappearance of cell nuclei alone. Thus the term "necrosis of the cornea" conveys the idea that the fixed corpuscles have been destroyed but that the lamellae, which are more resistant, are still living and are capable of being resuscitated by nuclear elements derived from a proliferation of cells at the limbus. Should the corneal lamellae be destroyed as well as the nuclear elements, there results a slough, in the sense that the entire area is dead and is cast off from the living parts by inflammatory reactions.

1. Fuchs, E.: Arch. f. Ophth. **77**:304, 1910.
2. Samuels, Bernard: Tr. Ophth. Soc. U. Kingdom **49**:421, 1929.

In considering the effects of toxins, one should note not only the location of the tumor in the globe but the location of the necrotic area within the tumor. In a mushroom-shaped sarcoma toxins from a necrotic focus bordering the sclera are likely to affect the sclera alone, whereas toxins from a necrotic focus in the head of the same tumor are apt to affect the ciliary body and iris, which are nearer to it. Toxins from a necrotic focus in the depth of a large sarcoma before they reach the surface probably lose some of their virulence by diffusing through layers of living tumor. Toxins may be held back by membranes that have formed on the surface of a tumor. A membrane familiar microscopically is the one that binds a degenerated retina to stretches of the inner surface of a sarcoma.

According to the degree of necrosis the specimens were grouped as follows:

Partially necrotic	60
Completely necrotic	24
Total	84

Among the sixty growths listed as partially necrotic were a number in which only a few living cells could be found. Usually the largest areas of necrosis were associated with the most extensive damage to the tissues of the globe. Of the twenty-four cases of completely necrotic tumors there were three in which live tumor cells were encountered in emissaria. In one case the extra-ocular nodule appeared to be as totally necrotic as the intra-ocular growth.

GENERAL EFFECT OF TOXINS ON THE VARIOUS TISSUES OF THE GLOBE

The globe is composed of tissues of widely different origin and structure. The retina is of exceedingly delicate texture, whereas the sclera is dense and resistant. Consequently there is a difference in the manner in which these two layers react when acted on by toxins. The iris often shows death of both nuclei and fibers, but the cornea ordinarily shows only disappearance of the fixed corpuscles. The pictures presented by globes that have suffered from necrosis differ according to the time that has elapsed since necrosis took place. In the first hours or days there may be but slight inflammatory reaction, whereas later the reaction may be severe. Necrosis does not manifest itself clinically; it is the reaction that is seen. Old cases of necrosis show connective tissue membranes on the internal surface of the uvea or in the tumor as a result of inflammatory reaction. Recent cases present no such membranes because there has not been time for them to develop.

The microscopic material was divided into two groups, in accordance with evidence that a long or a short period had ensued since necrosis occurred.

GROUP I: RECENT CASES (WITHOUT MEMBRANES)

There were fifty-eight cases in this group. In some of the eyes scarcely any inflammatory reaction was seen, whereas in others there was severe inflammation. When toxins from a highly necrotic sarcoma

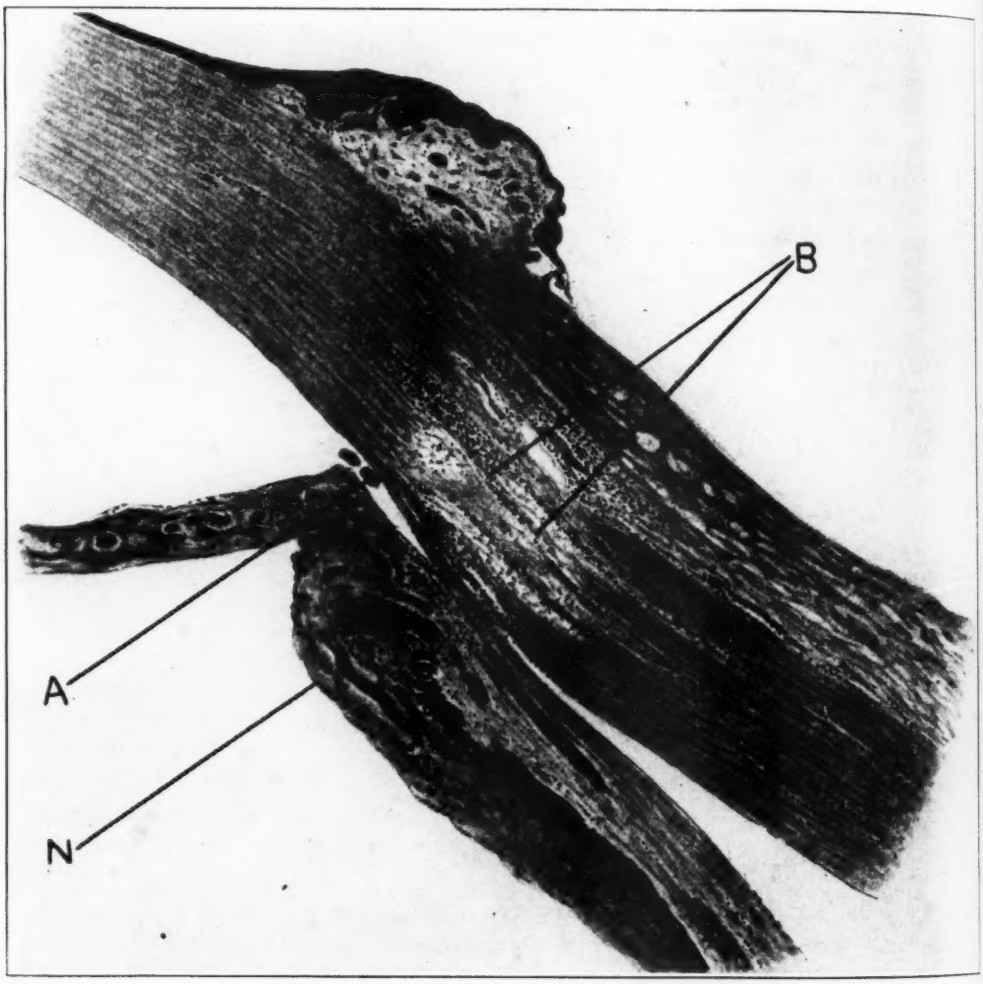

Fig. 1.—The root of the iris (*A*) was originally pressed against the pectinate ligament, from which it was separated later. The corneoscleral tissue (*B*) is heavily infiltrated and partly dissolved. Necrotic ciliary processes (*N*) are seen.

are set free in the interior of an eye they may cause such sudden and complete death of the tissues as to incapacitate them from reacting. There were instances in which the clinical histories mentioned that eyes were inflamed for a number of days before enucleation and yet microscopic examination revealed almost no evidence of reaction within the

globe. The inflammatory signs referred to were explained by the finding of lymphocytic infiltration in the sclera. These were examples of a reaction to toxins by tissue that was irritated but not destroyed.

The early effects of toxins on the individual parts of the eye were studied in the following order:

The Sclera.—A fully developed sarcoma replaces the perichoroidal lamellae and lies in contact with the sclera, to the lamina fusca of which it is commonly anchored by pervasion. An analysis of the location of

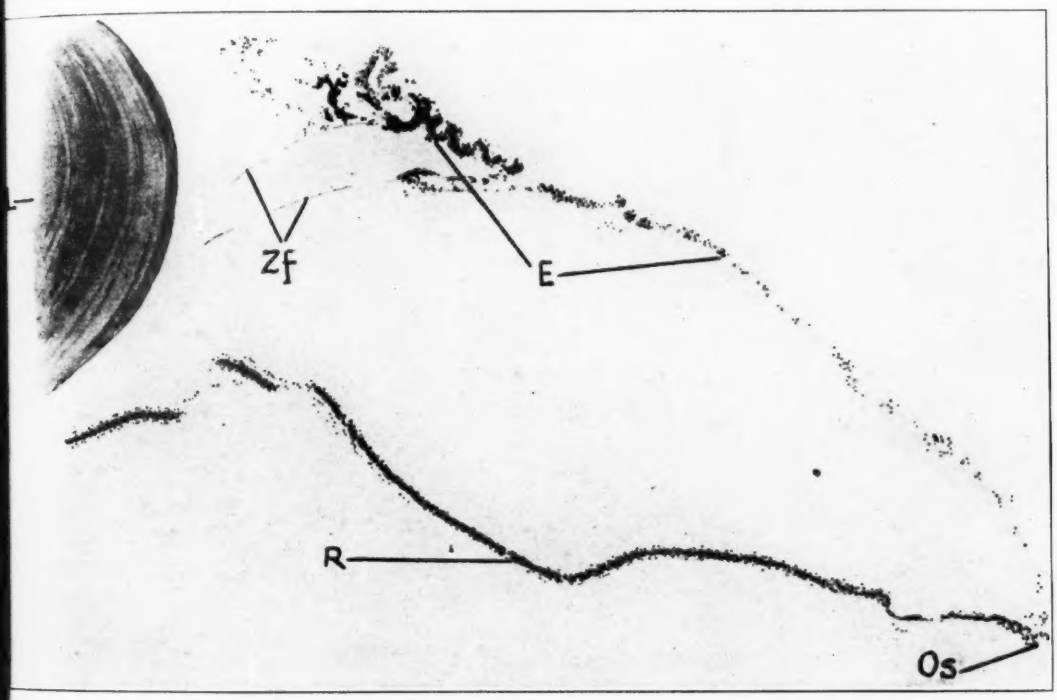

Fig. 2.—The lens (*L*) separated from the ciliary body and fell back into the vitreous cavity. The epithelium of the ciliary body (*E*) and the necrotic retina (*R*) were drawn back by the traction of the zonular fibers (*Zf*). The ora serrata is indicated by *Os*.

necrotic areas within the tumor revealed that they occurred almost as frequently on the side of the sclera as on that of the vitreous. The inner layers of the sclera showed areas of necrosis or of inflammation in thirty-three of the fifty-eight recent cases. In ten of these there was a parallel inflammation in the outer layers which was separated from the inflamed inner layers by an apparently normal scleral stroma. To explain this independent episcleritis, it was assumed that toxins had traveled through emissaria and in this way had reached the surface without having acted on the nuclei of the dense middle layers of the

sclera. Interstitial scleritis is rare under all circumstances, seemingly on account of the compact arrangement of the bundles of connective tissue and the scanty distribution of blood vessels among them. In three cases of totally necrotic tumors the adjacent sclera was liquefied, so virulent were the toxins. In eighteen cases the emissarium of one of the posterior ciliary arteries or nerves was invaded by tumor cells. In most of the eighteen, the extension of the tumor was confined to the inner half of the sclera. It was expected that tumor cells would be found in emissaria more frequently because the survey dealt with tumors of large size.

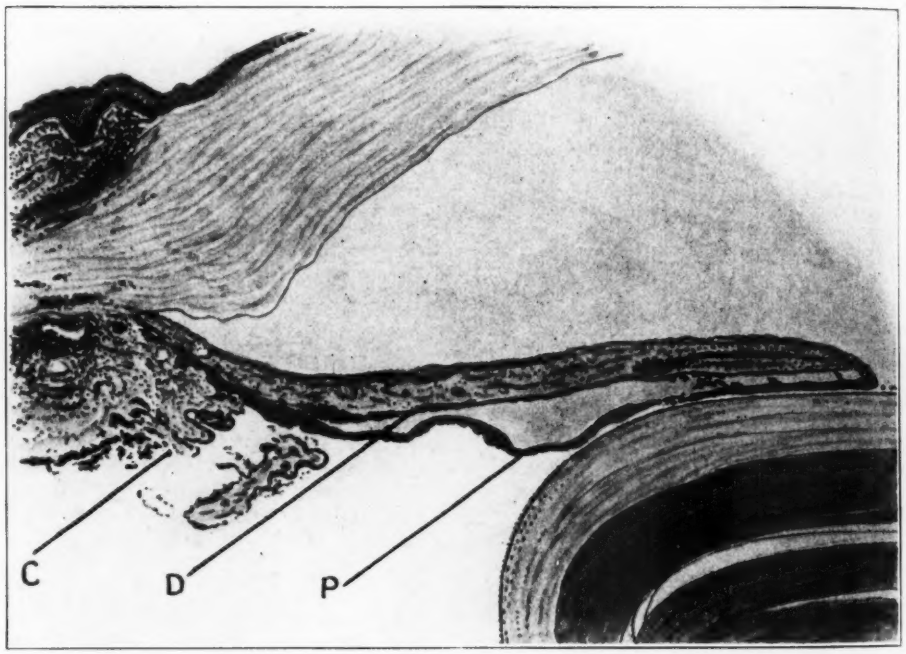

Fig. 3.—The pigment epithelium of the iris (*P*) was detached from the dilator muscle (*D*) by an albuminous fluid. The ciliary processes (*C*) are partly destroyed. The root of the iris is pressed against the cornea.

The Choroid.—Although the tumors here considered had their origin in the choroid, it was shown to be the least often and least heavily affected by toxins of all the structures of the globe. There is generally a narrow zone of lymphocytic infiltration in the choroid surrounding the base of a choroidal sarcoma, no matter how small, but this reaction may be the result of circulatory disturbance rather than of toxins. Toxins from a necrotic tumor reach the choroid in most instances in a roundabout way and not by continuity from the base of the tumor. When the necrotic focus is located at the inner surface of a tumor and there is complete detachment of the retina, toxins must first diffuse

through the fluid of the subretinal space, and then through the pigment epithelium, the lamina vitrea and the choriocapillaris before they reach the layer of large blood vessels. In many cases the pigment epithelium showed the effects of toxins by disappearance of the cement substance between the cells, which lost their hexagonal shape, became round and swollen and in order to find room heaped themselves on the lamina vitrea. Several times long stretches of the pigment epithelium were lifted bodily from the lamina vitrea by albuminous transudation from the choroid.

Necrosis of the stroma of the choroid manifested itself by edema and absence of nuclei. Usually the chromatophores had disappeared or had gathered in their processes and taken an oval or round shape.

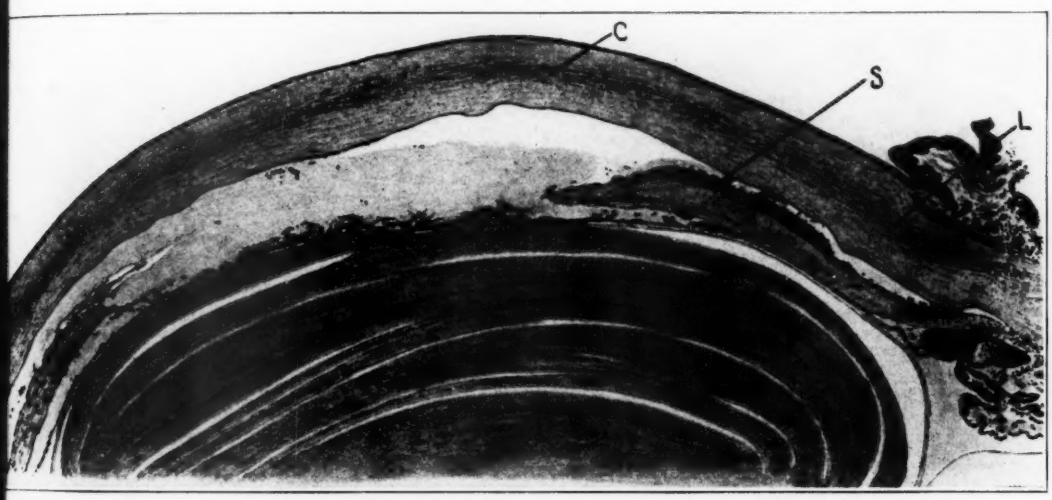

Fig. 4.—There is an infiltration in the middle layers of the cornea (*C*) and the limbus (*L*). The iris is completely necrotic, and its anterior surface (*S*) is lined by a heavy coating of pigment derived from disintegration of the pigment epithelium of the posterior surface.

No instance of hemorrhage into the stroma was seen. In one case toxins attacked the choroid on both sides. The retina was destroyed, and through a totally necrotic spot in the choroid toxins passed and spread along its external surface, so that both surfaces were covered with a layer of thickly packed pus cells.

The Retina.—The nuclei of the retina withstood the action of the toxins better than the supporting fibers, which were the first to disappear. The supporting fibers of the retina are so weblike that even before necrosis of a tumor begins they are apt to undergo cystic degeneration in areas where the retina is adherent to the tumor, apparently because of a disturbance of circulation. Large retinal vessels near the

optic nerve were observed in a number of cases, filled with blood and standing out in the vitreous cavity denuded of retinal tissue. Often the only nuclei in them that escaped destruction were those of the endothelial lining, which by being bathed in flowing blood were preserved. Occasionally a fine homogeneous strand floated about—a fragment of the membrana limitans interna of the retina. No doubt, in some cases the nerve fiber and ganglion cell layers had atrophied on account of the increased intra-ocular pressure which developed before the tumor became necrotic. The zone of the retina at the ora serrata proved vulnerable to the action of toxins. In a striking manner globe after globe showed sloughing of the retina in this zone, so that the remaining

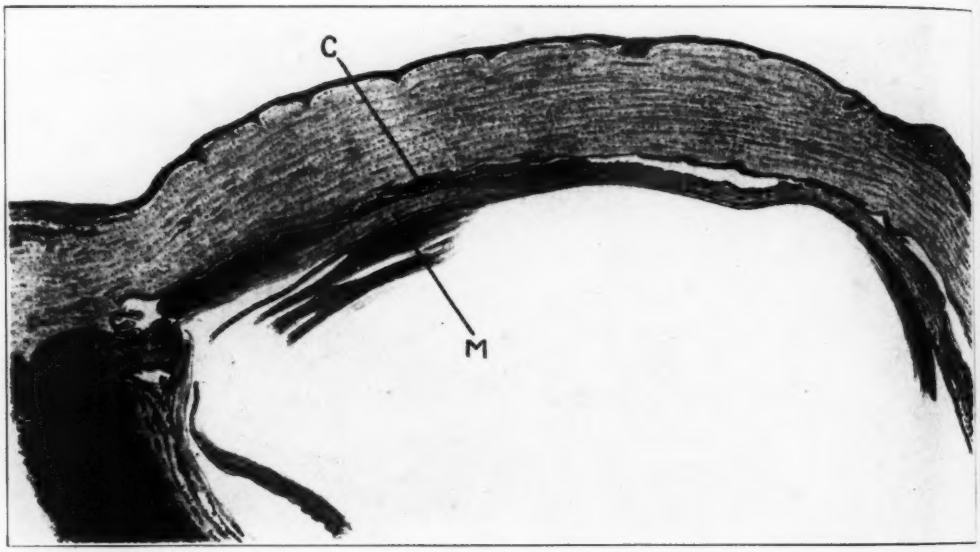

Fig. 5.—The flattening of the cornea and the wrinkling of Bowman's and Descemet's membranes are characteristic of an atrophic globe. The iris is represented by a dense membrane (*M*). The necrosis of the tumor was incomplete and cells (*C*) are now detaching Descemet's membrane.

portion folded on itself and fell back into the vitreous cavity. The retina was partially dissolved in all cases of incomplete necrosis of the tumor, and when the necrosis was extensive almost no vestige of the retina was visible.

The Ciliary Body.—The ciliary body suffered more frequently and more severely than any other division of the uveal tract, in spite of the fact that the majority of tumors were located in the posterior half of the globe. Toxins from a necrotic area at the surface of a tumor that extends far into the vitreous cavity may readily reach the ciliary body and iris by diffusion. The ciliary body lacks the protection on its inner surface that the vascular retina affords the choroid, and besides, the

lamina vitrea is here very thin and there is no capillary layer. In practically all cases in which toxins reached the inner surface of a tumor, the ciliary processes were damaged. Generally the damage was far more marked in the processes on the side of the tumor. When necrosis of a tumor was total or extensive the processes were found to have sloughed off, leaving the inner surface of the ciliary body smooth and even. Sometimes free blood outlined by pigment granules indicated the processes. Now and then the wall of a vessel engorged with blood was all of a process that survived. No traces of the nonpigmented epi-

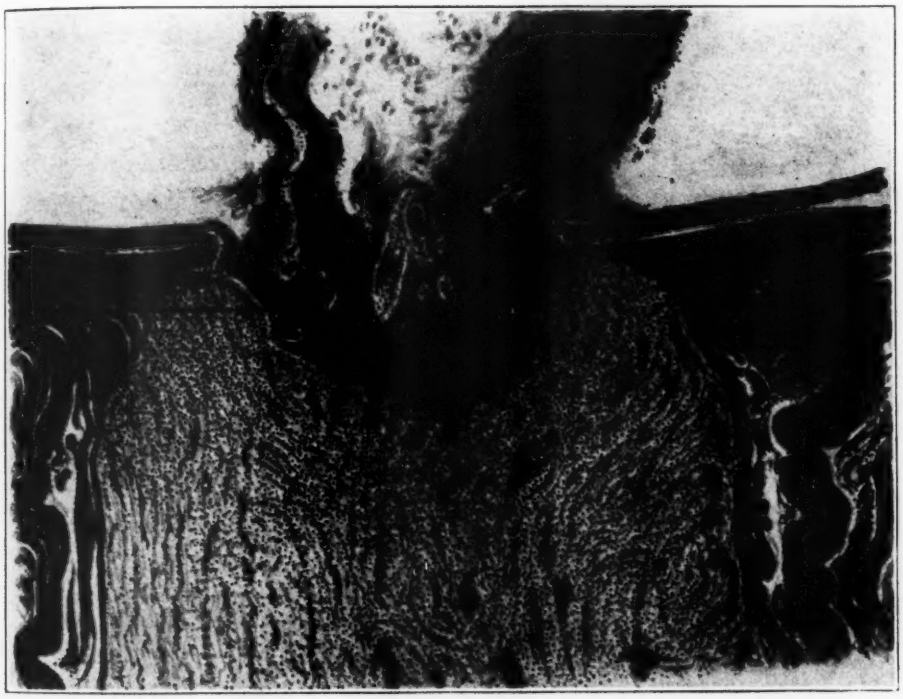

Fig. 6.—The glaucomatous excavation (*E*) is filled by necrotic papillary and retinal tissue permeated with blood. The inflammatory reaction extends far backward in the nerve trunk.

thelial layer were ever seen over a necrotic area. The pigment epithelium showed changes similar to those seen in this layer over necrotic areas of the choroid. The cells usually fell away from the surface individually. Groups of pigmented epithelial cells were occasionally seen preserved in bays between processes where there had been protection from direct action of toxins. The circular bundles of the ciliary muscle were often necrotic, but the longitudinal bundles were usually well preserved owing to protection afforded by the vascular layer on one side and the sclera on the other. One case was remarkable in that necrosis extended

through all the layers of the ciliary body and through the adjoining portion of the sclera. Small abscesses were present in the sclera, which was very rarefied. The inflammation and dehiscence are all the more remarkable when one considers how far away from the necrotic tumor this area of the sclera was. In another case the ciliary processes on the side of the tumor were spared, whereas those on the opposite side were necrotic. The lens had become dislocated and had lodged against

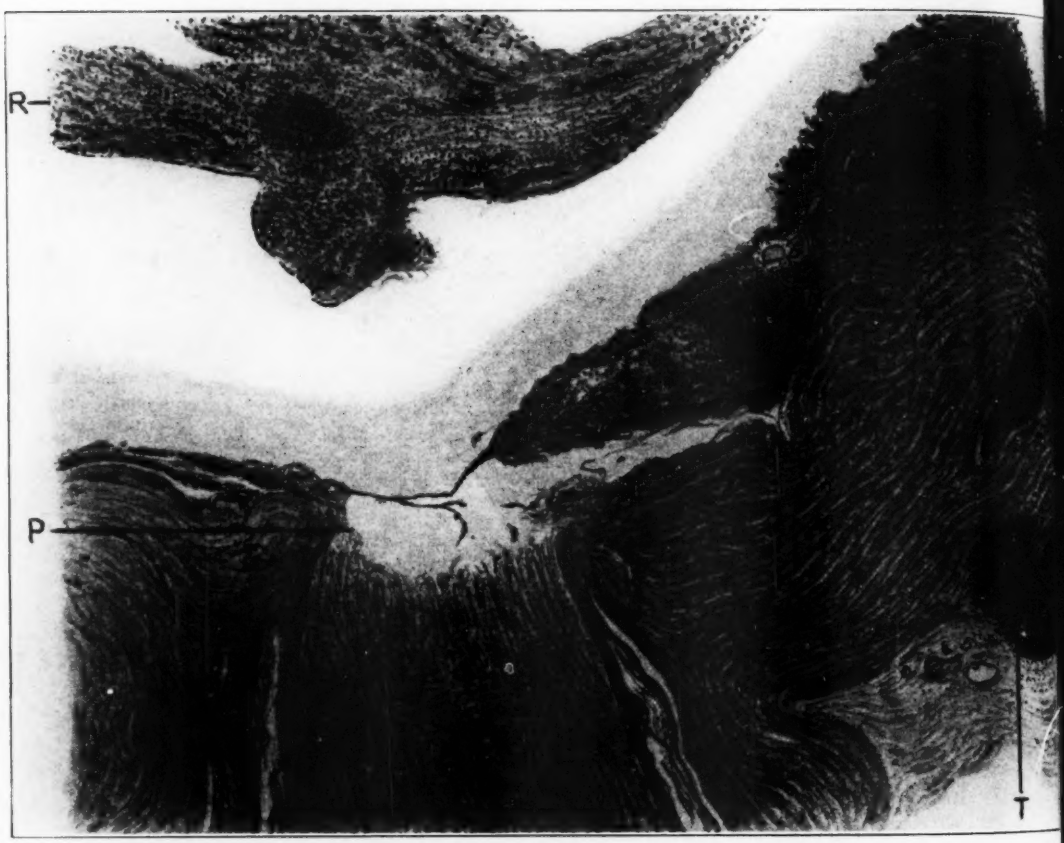

Fig. 7.—By the contraction of membranes the retina (*R*) was drawn forward, taking with it the entire necrotic papilla (*P*). *T* indicates a tumor perforating the sclera.

the ciliary processes on the side of the tumor, protecting them against the direct diffusion of the toxins.

The Iris.—When the ciliary body showed necrosis, the iris was almost sure to show it also. It was always more marked in the iris on the side that was nearer to the more necrotic ciliary processes. That the iris was so often necrotic when so great a distance intervened between it and the tumor was probably the result of several factors. The eyes

were nearly all glaucomatous. Acute fulminating glaucoma may of itself cause necrosis of the iris by suddenly compressing the blood vessels at the root of the iris. The necrosis of the iris observed in this series of cases of necrotic sarcoma was, as a rule, more extensive than that which one is accustomed to see in cases of ordinary glaucoma. Most likely the toxins acted in many instances on an iris already greatly damaged. There were two eyes showing unusually broad adhesions at the angle

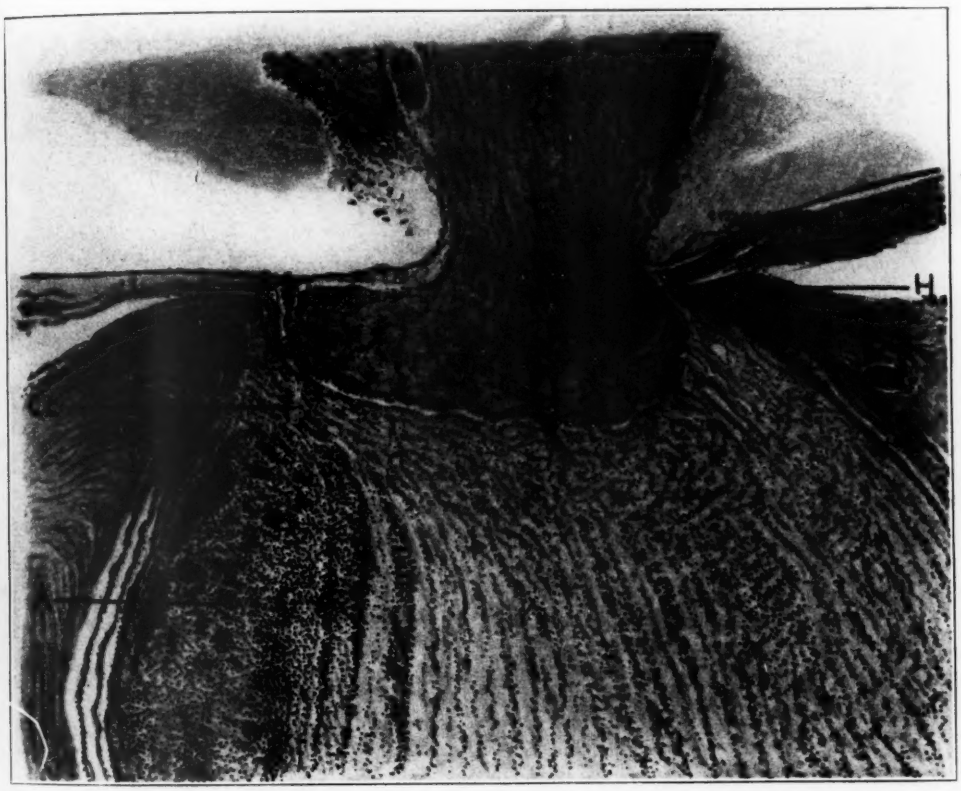

Fig. 8.—The lamina cribrosa (*Lc*) is pushed back, and the space anterior to it is filled with papillary and retinal tissue, in the midst of which is a hemorrhage (*H*). The trunk of the nerve shows a large abscess (*A*), which contains blood pigment.

in which the necrosis of the iris was believed to be due to glaucoma alone. The tumors were near the posterior pole, and the necrosis in them was partial. The retinas were completely detached and pressed the lenses forward. The fact that the ciliary bodies showed no necrosis indicated that toxins had not diffused far forward. In some severe cases of necrosis long strips of the pigment epithelium fell away from the iris. In several of the cases in which the pupillary margin of the iris was free, sheets of dead epithelial cells had floated forward and

were found deposited on the anterior surface of the iris. It was remarkable not only how often the pupillary margin was free but how many times it pointed forward owing to edema in the posterior layers of the stroma. The pigmented cells showed a remarkable tendency to migrate into the stroma as if to escape toxins that were pursuing them from the posterior chamber. In the midst of a necrotic stroma thick walls of vessels were often preserved as pinkish tubes engorged with blood. Many times free blood was encountered in the edematous stroma. As a rule the pupillary zone of the iris suffered more than the

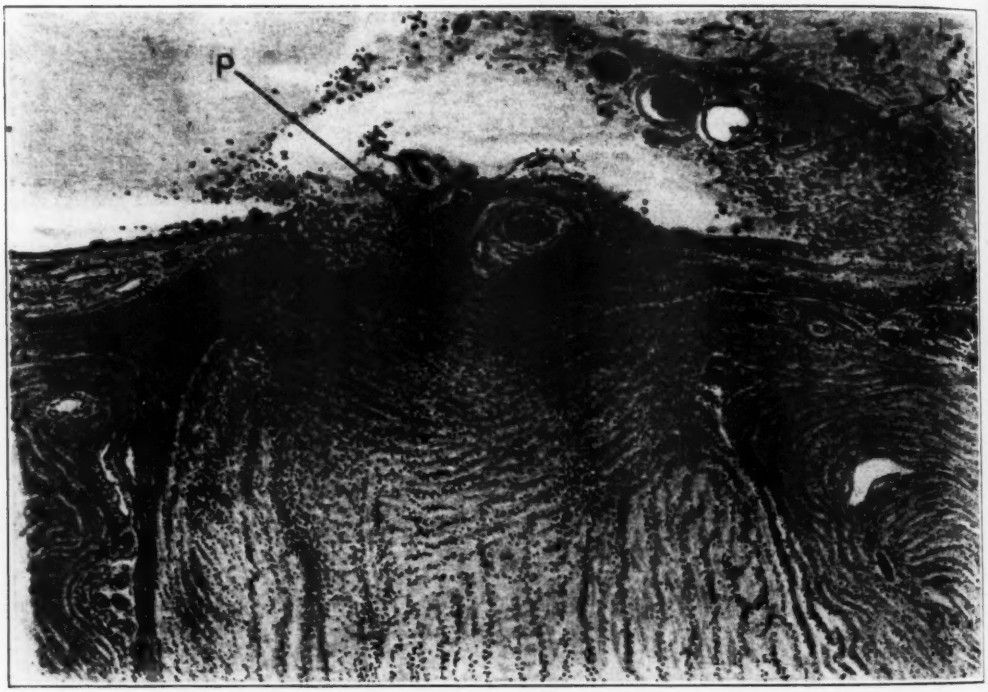

Fig. 9.—As a result of intense necrosis the papilla (*P*) was destroyed and the retina (*R*) separated from it. From the necrotic area phagocytes filled with pigment invaded the nerve, collecting especially at the periphery.

ciliary zone, possibly because it is the farthest away from the blood supply and because it was freely exposed to toxins as they passed forward into the anterior chamber.

The Anterior Chamber.—(*a*) The Depth: A shallow anterior chamber was found in the majority of the cases. In eight eyes the anterior chamber was totally abolished. In two it was deep; one globe was atrophic, and in the other the lens was luxated. A broad peripheral anterior synechia was found in all of the eighty-four cases except five. It is of interest to review these exceptional cases. In the first there was extensive necrosis of the tumor, but only limited necrosis in the

uveal tract because the necrosis was centrally located in the tumor. In the second case a paracentesis had been performed. The eye was not painful before the operation but within a few hours following it violent inflammation came on. Large areas of necrosis and hemorrhages were found in the tumor, and the vitreous was permeated with blood. In the third case there was partial necrosis of the tumor, with limited necrosis in the tissues of the eye. The fourth and fifth cases were similar to the third. These were not typical cases in that neither the tumor nor the

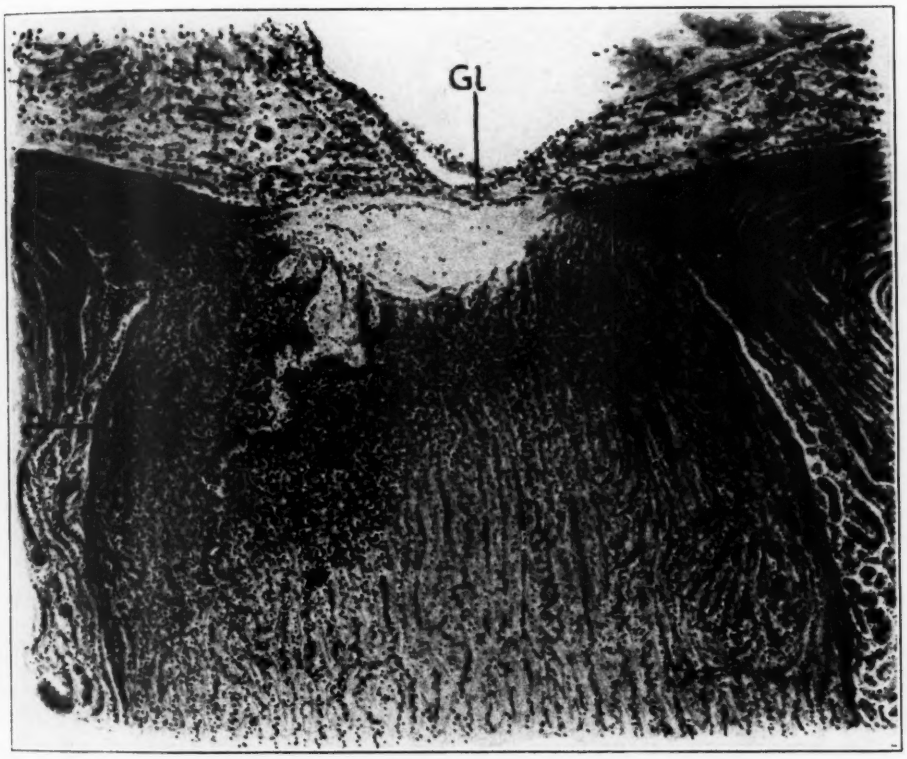

Fig. 10.—The glaucomatous excavation was cut tangentially and is bridged by a filament of glia (*Gl*). To the right the choroid is necrotic but recognizable; to the left the choroid and retina are fused on account of necrosis. Portions of the cribriform fascia and of the nerve substance are necrotic and are invaded by phagocytes packed with pigment. These cells are strikingly collected near the pia (*P*).

tissues revealed much necrosis. However, the absence of adhesions in five cases in which the necrosis was slight and the presence of a broad peripheral synechia in cases in which necrosis was great led to the conclusion that toxins, by irritating the iris, were a factor in the formation of adhesions that were usually broader than those seen in cases of primary glaucoma of long standing.

(*b*) The Contents: In seven cases the anterior chamber was almost filled with a combination of blood and pigment granules. In eight cases it was filled with blood alone. In these fifteen cases there was extensive necrosis of most of the structures of the globe. Ordinarily the anterior chamber contained a considerable number of pigment granules. There were four cases of fibrinous exudate in the meshes of which there was a scattering of pigment granules and red blood cells. The fibrin seemed to protect the iris from toxins, as the anterior layers were well preserved in these cases. Pure hypopyon was present in but one case. This was in an eye the contents of which were largely replaced by a necrotic

Fig. 11.—This section is from the same eye as that shown in figure 5. There is direct propagation of pigmented tumor cells (*Ptc*) into the nerve from a necrotic sarcoma (*Ns*).

sarcoma, in the midst of which there was pus. The rarity of hypopyon in cases of necrotic sarcoma is worthy of note. In cases of serpiginous ulcer of the cornea toxins diffuse from the external surface into the anterior and posterior chambers and by irritating the iris and ciliary body cause migration of leukocytes into the aqueous (hypopyon). When in a case of necrotic sarcoma the toxins, which may be even more noxious than those from bacteria, attack the ciliary body and iris, they may damage these tissues so severely that they have not the power to throw out an exudate, which explains the rarity of hypopyon in association with necrotic intra-ocular sarcomas.

The Cornea.—The cornea, although farthest removed of all the tissues from a choroidal sarcoma, was almost as frequently necrotic as the iris, but never to the same extent. A markedly necrotic iris was usually accompanied by a necrotic area in the cornea. The necrosis involved the layers of the posterior third of the central area. Corresponding to

Fig. 12.—As a result of toxins produced by complete necrosis of a large tumor (*T*) pus formed along the inner surface of the sclera, resulting in a perforation (*P*). The retina is everywhere necrotic and has fallen to pieces at *R*. The lens (*L*) was dislocated by the ciliary portion of the tumor. The anterior chamber is filled with pigment and blood.

the necrotic area in the stroma, there was absence of endothelium. The epithelium on the anterior surface was most often little affected. Impressive was one case in which the nuclei had disappeared throughout

the entire thickness of the cornea and yet the overlying epithelium appeared to be normal. There was one case of keratitis bullosa, and there was another case in which the corneal epithelium was markedly edematous. Both eyes were severely damaged by necrosis. Favoring central necrosis of the cornea are, first, the inactivity of the lymph flow in this area as compared with that at the periphery near the loop of blood vessels and, second, the direct exposure of this area to the toxins as they are carried forward in the aqueous through the pupil. The cornea may also become necrotic in cases of acute glaucoma with high tension. In seven eyes in which almost every structure had succumbed to necrosis, the cornea showed a heavy infiltration in the periphery and absence of nuclei in the center. The picture was that of a ring abscess as it is seen in panophthalmitis.

The Vitreous.—Liquefaction of the vitreous was noted whenever necrosis of the tumor was considerable. The fluid that replaced the vitreous body was albuminous, often containing pigment granules and in addition free blood derived from sloughing ciliary processes or from necrotic areas of the tumor. In one case extravasation of blood was so great that a diffuse lymphocytic infiltration in the choroid was believed to have been caused by toxins of degenerated red blood cells rather than by toxins of the necrotic tumor. Verhoeff [3] has pointed out that hemorrhages in blind eyes are most suggestive of the presence of a sarcoma.

The Zonular Fibers.—The zonular fibers were more or less dissolved by toxins in most cases. In one of the two globes that had a deep anterior chamber the zonular fibers, together with the pigment epithelium of the flat part of the ciliary body, fell away and with the lens sank back in the fluid of the vitreous cavity. The fibers and epithelium were probably pushed away from their attachment by fluid transuded from the vascular layer of the ciliary body. Sometimes pigment granules disposed in long chains floated near the ciliary body. They were believed to be clinging to thin zonular fibers or strands of the anterior limiting layer of the vitreous, the granules being visible whereas the structures to which they clung were invisible.

The Lens.—The lens substance showed a tendency to swell in severe cases of necrosis. Several times it became almost round, and once it was so large and tumescent that the slide bore the legend "giant lens." In many eyes the cortical layers appeared to be liquefied, the nucleus being preserved because of its density, as these were mostly eyes of elderly people. The epithelial cells lining the posterior surface of the anterior capsule were more often missing than those at the equator in spite of the fact that the latter were in closer proximity to the tumor.

3. Verhoeff, F. H.: Tr. Am. Ophth. Soc. **30**:100, 1932.

There were cases of severe necrosis in which no nuclei were seen in the lens, indicating complete necrosis. At times the epithelial cells on the side of the tumor were missing. In the two cases in which the lens fell back in the vitreous the epithelium was living, so that it seemed to make little difference to the contents whether the lens was luxated or not. In three cases of severe necrosis the posterior capsule ruptured spontaneously.

The Optic Nerve.—A pathologic excavation was present in a majority of the cases. The excavation was usually not deep, and it was almost always less deep than one would expect in comparison with the

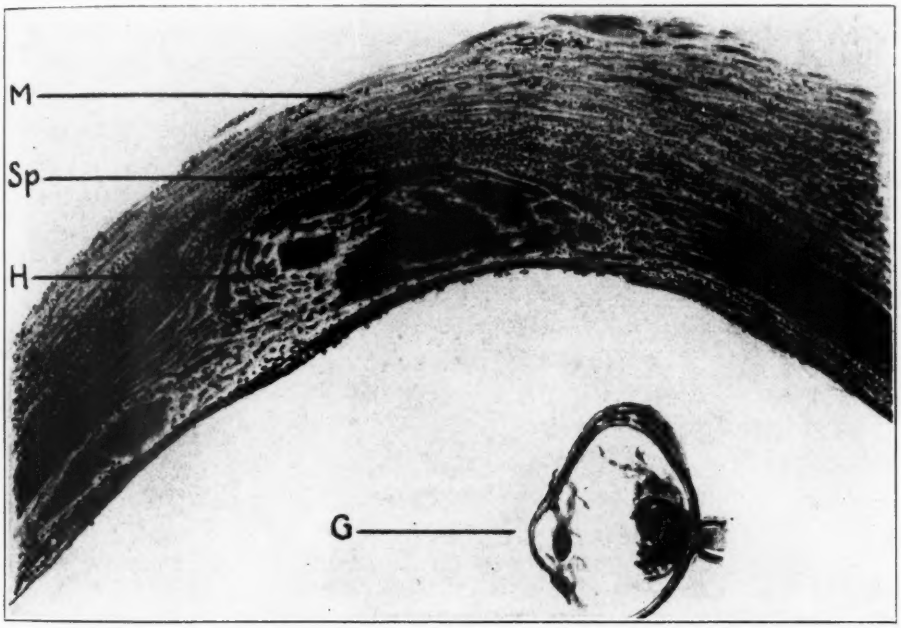

Fig. 13.—There has been spontaneous perforation of the sclera (*Sp*) as a result of inflammatory reaction. *H* indicates a hemorrhage, and *M*, a membrane enveloping the seat of the perforation. The globe (*G*) did not atrophy.

broad adhesion at the angle of the anterior chamber. Total necrosis of the papilla was frequently observed in connection with total necrosis of the retina. The papilla was converted into a faintly staining weblike tissue, which often held a few nuclei from the columns of nuclei. In the midst of a necrotic papilla the walls of the central retinal vessels were more than once preserved, engorged with blood. In two instances necrotic plugs that had filled excavations were found suspended in the vitreous cavity, having been lifted from the cribriform fascia by membranes in the vitreous. That such a pathologic feat happened was proof that the nerve fibers and blood vessels had lost intimate connection with the cribriform fascia.

Extravasations of blood occurred in the papilla, and in a few cases hemorrhages were seen in the nerve at a considerable distance posterior to the cribriform fascia. Here they were nearly always found in the nerve fiber layers nearest to the pial sheath. Generally the blood vessels of the nerve were dilated and heavily engorged. In four cases of very severe intra-ocular necrosis there was a large abscess in the nerve posterior to the cribriform fascia, located near the pial sheath and on the side of the tumor. Abscesses in the optic nerve are rare, and their presence was a striking demonstration of the distance to which toxins of a necrotic sarcoma may be transported by the fluids.

A number of factors appeared to play a rôle in the formation of the pigment that was seen in the papilla, in the cribriform fascia and even farther back in the nerve. It is certain that the pigment could seldom be ascribed to an extension of the tumor into the nerve head. In only three of the eighty-four cases were tumor cells encountered in the substance of the nerve, in spite of the fact that there were fourteen circumpapillary tumors. In two of the three cases tumor cells first invaded the detached retina at the margin of the nerve and from the retina found their way into the papilla. In the third case the extension was along blood vessels of the sclera into the intervaginal space. In none of the three cases had tumor cells penetrated much beyond the posterior level of the cribriform fascia. There was a definite relationship between the pigment in the nerve and the necrosis of the sarcoma in that the pigment was scarcely ever seen unless the tumor was exceedingly necrotic; nor were abscesses or hemorrhages found in the nerve unless the tumor was very necrotic. Once a large accumulation of pigment granules on the nerve head came directly from an extensive blistering and breaking up of the pigment epithelium of the choroid after the retina had been dissolved.

From the anterior surface of the nerve, pigment granules may be carried backward by the lymph stream, either free or in the body of phagocytes, and be deposited in the tissues of the nerve. Several times pigment granules were found in groups of branched cells in the cribriform fascia or immediately back of it, and always near the pial sheath. They were not chromatophores, which are sometimes seen normally in the sclera and cribriform fascia. They were too darkly pigmented and too densely disposed; moreover, there was no pigment elsewhere in the sclera or cribriform fascia. They gave the impression of being cells that had not created the pigment they contained but had taken it up. At times there was no doubt that the pigment in the nerve substance was of hematogenous origin, as the granules were large and irregular and olive brown and were found in the midst of disintegrating red blood cells. It is possible that in former times pigment in the nerve in cases of intra-ocular sarcoma was interpreted as due to living tumor

cells and so the practice grew of excising as long a strip of the nerve as possible in all cases. According to the specimens there is no anatomic basis for this surgical procedure unless the sarcoma is located near the nerve head.

GROUP II: OLD CASES (WITH MEMBRANES)

There were twenty-six globes which had passed through the inflammatory period and at the time of enucleation were in a state of quies-

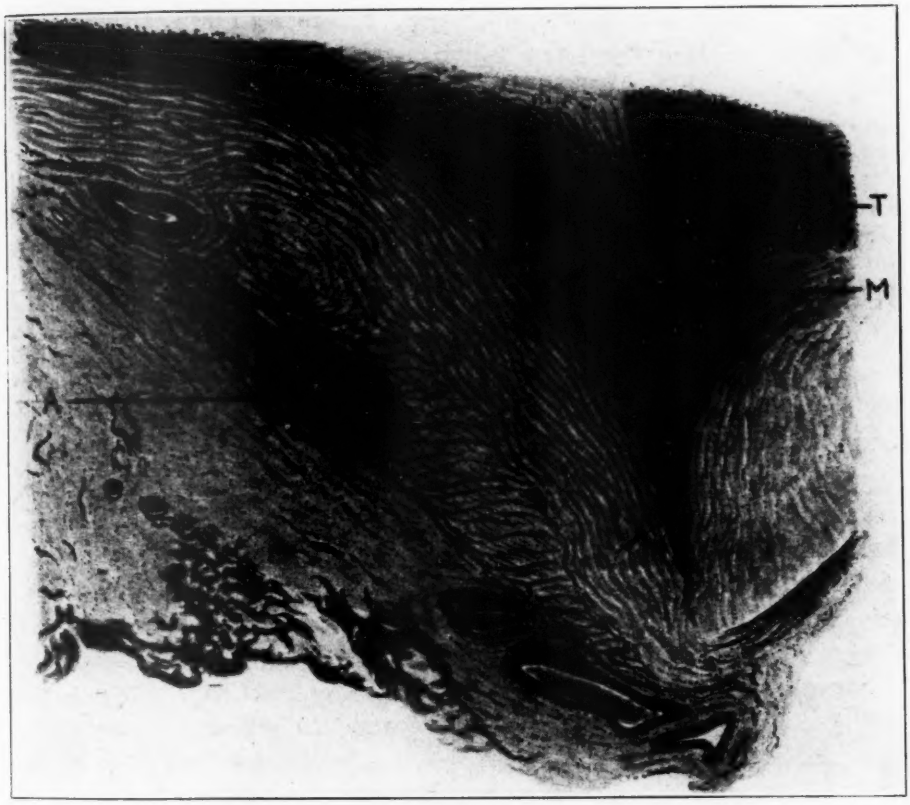

Fig. 14.—*A* indicates a hypertrophic artery on the external surface of the sclera. The tumor (*T*) is traversed by broad membranes (*M*).

cence. Fourteen were smaller than normal, and two were phthisical. The remaining ten retained their normal size. The connective tissue membranes that characterized the group of old cases were laid down on the uvea or in the tumor. The membranes were the result of organization of blood or of exudate thrown out by those parts of the uvea and sclera that had been spared, from which sources fibroblasts and capillaries invaded the blood or exudate, replacing it with a membrane. The membranes did not appear to be so dense and so contractile as those that replace ordinary purulent exudates, which may be a reason why more

of the globes were not shrunken. It was not always easy to distinguish cells of a new membrane from those of a recurrent sarcoma. However, the fibroblasts were more spindle-shaped and took a deeper blue stain and were not so succulent looking as the tumor cells.

The following sequelae of necrosis and of inflammation were noted in the various spaces and tissues of the globe:

The Vitreous Cavity.—Broad bands of connective tissue often traversed the vitreous space, in the midst of which there were frequently islands of tumor cells. At times bands of connective tissue enclosed cavities containing débris of tumor cells and red blood corpuscles, together with cholesterol spaces. In several cases a diagnosis of an organized blood clot in the vitreous cavity might have been made had not a few living tumor cells been discovered. In many instances bands of connective tissue swept directly from the sclera into the midst of a necrotic sarcoma. Probably these bands were derived from the walls of blood vessels in the emissaria.

The Lens.—In most of the old cases the lens had fallen far back in the vitreous space. The lamellar layers were commonly liquefied and in several instances were calcareous. In one case there was an anterior polar cataract that lay under a thin membrane that stretched across the pupil.

The Anterior Chamber.—The old cases were remarkable for an absence of organized exudate in the anterior chamber, which corresponded to the infrequency of hypopyon recorded in the recent cases. Seldom was there any pigment, because in the course of time the granules probably had been removed by phagocytes. In one anterior chamber there was a mass of nonpigmented epithelial cells lying on the iris. They were cells that had fallen away from Descemet's membrane as a result of overproliferation of epithelium in an endeavor to cover a denuded area.

The Choroid.—In several instances the choroid was covered by peculiar laminated membranes, with streaks of pigment between the layers. These membranes were laid down by the pigment epithelium in cases in which it had been irritated by toxins. Underlying the membranes traces of the lamina vitrea could be seen. The membranes did not stain red by the van Gieson method, as does connective tissue. Usually there were ordinary connective tissue membranes overlying the choroid, often containing pigment granules. Once a membrane entirely encapsulated a necrotic tumor. There were several cases in which portions of the membranes were transformed into bone. As an exception, in one globe the stroma of the choroid was replaced over short stretches by connective tissue, which possibly represented organized hemorrhages.

The Ciliary Body.—Membranes often covered the internal surface of the ciliary body, but there was seldom proliferation of the epithelial layers, such as often occurs in chronic iridocyclitis. The membranes formed after sloughing of the epithelial layers and ciliary processes had taken place, and therefore they blended with the vascular layer of the ciliary body. Generally some of the longitudinal fibers of the ciliary muscle were left, but in several instances these were replaced by a layer of connective tissue. In four cases the entire circumlental space was filled with organized exudate rich in pigment granules. In these there had been produced great irritation in the ciliary body, but not necrosis, resulting in a thick cyclitic membrane.

The Iris.—The appearance of the iris in the old cases varied according to whether or not the iris was adherent to the lens. When adherent it was usually stretched into a thin, homogeneous layer containing much pigment. When not adherent it was shrunken to a mere stump. Such shrinkage of the iris may occur within a few days in cases of acute fulminating glaucoma. Rapid shrinkage may be interpreted to be the result of necrosis and not of atrophy.

The Cornea.—Comparatively seldom did the cornea show many changes. In a few cases there were deep blood vessels, and in others there was wrinkling which corresponded to the general folding of the sclera. Of great interest was a case in which Bowman's and Descemet's membranes were unimpaired whereas a considerable portion of the parenchyma was replaced by cellular, wavy connective tissue. Possibly in this instance many lamellae had been destroyed by necrosis and were replaced by ordinary connective tissue. There was one example of invasion of the cornea by tumor cells. Descemet's membrane offers great resistance to tumor cells. Finally, there was a case in which the cornea had been destroyed by panophthalmitis in connection with destruction of most of the other structures of the globe. The uncommonness of scar tissue in the cornea in the old cases may be explained by recalling that the necrosis referred to in the recent cases usually consisted of death of the nuclei only.

The Sclera.—One of the most interesting facts brought out by the survey was the rarity with which sarcoma cells pervaded the entire thickness of the sclera. There was but one case in which tumor cells were found between all the layers of the sclera. There were seven cases in which it could be said definitely that tumor cells had reached the surface through large openings that had been formed by necrosis in the sclera. The extension of cells through such openings must have developed long after necrosis occurred, because before the sclera became necrotic the neighboring parts of the tumor were dead. By the time new tumor cells could have grown, membranes had formed to cover

the defect. By propagating through new-formed tissue, less resistant than the original sclera, these seven tumors found their way to the outside of the globe. In one case a thick membrane that bridged a large defect in the sclera became ectatic through pressure of tumor cells, so that there developed a large extra-ocular nodule entirely covered by a membrane that was attached to the sclera. In another case, in a highly myopic eye, the sclera became thin and greatly ectatic over a necrotic sarcoma located near the posterior pole.

At times broad membranes of connective tissue encircled the sclera. They were the result of orbital cellulitis. Masses of connective tissue not infrequently clung to the sclera, which at first glance might have been mistaken for an extra-ocular extension of a sarcoma. E. Fuchs once reported a case in which after the excision of a globe a mass was felt in the orbit which was believed to be an extension of an intra-ocular sarcoma. Exenteration of the orbit was performed. The mass proved to be new-formed connective tissue. An extra-ocular nodule that is the product of extension through an emissarium probably grows more rapidly than one resulting from extension through an opening formed by necrosis, because in the former there are no inflammatory membranes to impede growth.

SUMMARY

A summary of the degree of necrosis in the tumors and in the tissues is given in tables 1 and 2.

TABLE 1.—*Degree of Necrosis in Tumors*

	Recent Cases	Old Cases
Total necrosis	15	5
Only a few living tumor cells	14	3
Partial necrosis	26	8
Intra-ocular tumor totally necrotic (recent cases)	3	..
Intra-ocular tumor alive (old case)	..	1
Newly growing tumor (old cases)	..	9
Total	58	26

TABLE 2.—*Degree of Necrosis in Tissues* *

	Iris	Ciliary Processes	Ciliary Body	Choroid	Retina	Lens
Extensive	23	27	16	12	26	18
Limited	12	7	2	21	7	5
Absent	4	18	31	19	19	23

* Only recent cases are included.

CLINICAL ASPECTS

Every period of life from infancy to extreme old age was represented in the fifty cases in which the ages were given. In nine the patient was 30 or under.

SUBJECTIVE SYMPTOMS

Visual Disturbance.—The histories brought out that the period of visual disturbance was generally from one to two years. Only six patients knew of complete blindness in the sarcomatous eye. Most of them spoke of gradually failing vision.

Inflammation and Pain.—These symptoms were almost invariably said to have come on suddenly. So severe and intractable were they that most of the patients could bear them not longer than a few weeks before submitting to enucleation.

OBJECTIVE SYMPTOMS (RECENT AND OLD CASES)

A review of the statements that were made in the histories concerning those parts of the globe that lend themselves to clinical examination follows.

The Cornea.—The changes in the cornea are summarized in table 3. The dulness of the cornea may have been caused by an increase of ten-

TABLE 3.—*Changes in the Cornea*

	Number of Cases
Dulness	20
Ring abscess	2
Vesicular keratitis with pigment on Descemet's membrane	1
Pannus	1
Gray stripes	1
Changes not recorded	59
Total	84

sion in many instances. In one of the two cases of ring abscess microscopic examination showed a spherical tumor that filled almost the entire vitreous cavity. It was completely necrotic, as was almost the entire uveal tract, and in addition there was an inflammatory focus in the optic nerve back of the cribriform fascia. The tumor had a broad base on the sclera, and as the toxins diffused there was set up an inflammation that was spoken of as intense scleritis. The globe had been inflamed for nine days. The other eye with a ring abscess showed little scleritis. The sarcoma in this eye was mushroom-shaped. The intra-ocular nodule had become completely necrotic, and as the broad infiltrating base was but slightly necrotic, it served to protect the sclera from toxins. This eye had been inflamed for eight days, and there had been no vision in it for one year. The eye with vesicular keratitis had been blind for eighteen months and was painful for eight days. Under the microscope there was found total necrosis of the ciliary processes and of the iris, the pupil being free. The posterior layers of the cornea showed absence of nuclei as far as the limbus. The anterior layers were heavily infiltrated with leukocytes. The epithelium was missing. This must have been a case of sudden necrosis of the head of a large tumor, because the

ciliary processes had sloughed and the pigment epithelium was detached from the iris. The layers of the tumor bordering the sclera were well preserved, so that they protected the sclera from toxins. The eye with pannus had been blind for three years. The tension was high. There was hypopyon. A completely necrotic tumor occupied almost the entire vitreous cavity. Curiously, the toxins from so large a decayed mass had caused but incomplete necrosis of the ciliary body and iris, so that they were able to react and throw out an exudate.

The Anterior Chamber.—The clinical finding of a shallow anterior chamber in fourteen and abolition of the chamber in one of nineteen (table 4) cases was in keeping with the results of pathologico-anatomic examination, which in most instances revealed a shallow anterior chamber and a broad peripheral anterior adhesion. Histories were available for six of the eleven cases in which blood was found in the anterior chamber microscopically either alone or combined with pus and pigment. In each

TABLE 4.—*The Anterior Chamber*

Depth		Contents	
Normal	1	Hyphemia	7
Deep	3	Hypopyon	1
Abolished	1	Blood and pus	1
Shallow	14	Pus and pigment	2
Not recorded	65	Not recorded	73
Total	84	Total	84

instance the inflammation came on suddenly and was violent. The necrosis in these eyes proved to be exceptionally severe. Acute and severe inflammation may be said to go hand in hand with hyphemia.

The eye listed as showing hypopyon had exhibited failing vision for four months, and for three weeks it had been injected and painful. The cornea was described as "steamy." Microscopically there was a large amount of blood in one angle of the anterior chamber, but no pus cells were found. The periphery of the cornea was infiltrated with lymphocytes. Seen through a steamy epithelium and infiltrated stroma, the blood may have appeared as a yellow content. The iris, the ciliary body and the retina were highly necrotic, and there was blood in the vitreous. The lens had fallen back. The toxins came from a mushroom-shaped sarcoma, the head of which was totally necrotic and the base partially so. There was internal scleritis confined to the side of the tumor. There was an inflammatory area with much pigment in the optic nerve on the side of the tumor, posterior to the cribriform fascia.

Of the seven cases of hyphemia, in one the condition was described as "a black content" in the anterior chamber. Microscopically this proved to be a mixture of blood and pigment in almost equal amounts. It was in an eye that had been blind for five months and inflamed for five

days. There was a large spherical tumor that was necrotic. The anterior surface and one side of the tumor were covered by a membrane which probably kept back the toxins to such an extent that only the ciliary processes and iris were necrotic. The flat part of the ciliary body protected the sclera, so that there were few signs of scleral infiltration in the anterior half of the eye, whereas posteriorly a heavy membrane surrounded the sclera over the area on which the necrotic tumor sat. The optic nerve in this case had a deep glaucomatous excavation, notwithstanding that the adhesion at the angle of the anterior chamber was no broader than in many eyes that had less excavation. Apparently increased tension had preceded necrosis by a considerable period.

The eye in which the anterior chamber was described clinically as being abolished had a swollen necrotic iris, containing many hemorrhages, in contact with the cornea. The cornea showed necrosis of the posterior central layers. The ciliary processes were atrophic but not necrotic. The lens was pressed forward by a completely detached and partially necrotic retina. The tumor was near the posterior pole, and only small parts were necrotic. The necrosis of the iris, cornea and lens was believed to be due to compression from glaucoma and not to absorption of toxins; otherwise the ciliary body would have exhibited necrosis. The eye had been blind for one year and inflamed for eight days.

The Iris.—Frequently the histories mentioned iritis or iridocyclitis, but there was no indication that necrosis of the iris was suspected. However, one may suspect necrosis when one finds a greatly swollen, indefinitely colored iris showing hemorrhagic spots, especially if the pupillary margin is free. Again, in the clinical charts, the iris was several times referred to as having a black coating on its anterior surface, and once a patch of "black pigment" was seen. These were cases in which the sections showed a free pupillary margin, which permitted pigment to be carried forward from the posterior chamber and deposited on the anterior surface of the iris. In one case the iris was described as atrophic, and microscopic examination confirmed this and revealed small necrotic areas which were all the more marked because, having no nuclei, they appeared as almost empty spaces in very cellular tissue. The tumor was far back and contained comparatively small areas of central necrosis. The eye had been blind for six months and inflamed for eight days. The atrophy of the iris was considered to be due to glaucoma, and the necrotic areas in it to be due to toxins, as the ciliary processes showed necrosis with hemorrhages.

The Lens.—There was clinical mention of luxation in the case of a woman, aged 46. Vision began to fail during pregnancy, three and a half years before enucleation. Nine months later the eye became

totally blind. Absolute glaucoma developed. The lens was described as cataractous and as having fallen back, and turned so that the equator of the lens on one side touched the pupillary margin of the iris. Microscopic examination showed an iris shrunken to a mere stump of vascular connective tissue. The anterior surface of the stump was entirely covered by pathologic ectropion of the uvea. From one side of the pupillary border a membrane extended to the corresponding side of the lens. On the opposite side the shrunken lens was free in the midst of bloody fluid. The retina had disappeared. A spherical sarcoma occupied one half of the vitreous cavity. Dense membranes were present in the vitreous, enclosing large areas of blood as well as a few islands of tumor cells. The case was instructive because it appeared that a process of decay of masses of tumor cells and their replacement by connective tissue had been going on for many months. There was no evidence of extra-ocular extension.

The Retina.—In one case it was stated that the sight had not been good for one year and that for six months the retina was detached. Six days before enucleation the eye became extremely painful. There proved to be partial necrosis of the tumor, as well as of the ciliary processes, iris, lens and cornea. The sarcoma was a flat, infiltrating one at the posterior pole, and the retina was completely detached and largely necrotic. It is likely that the retina would have suffered total necrosis had it not been protected by an immense amount of subretinal fluid. In another case it was stated that the eye was blind for eight weeks and that glaucoma and detachment of the retina were present. Microscopically a spherical sarcoma sprang from the posterior segment and extended forward almost to the back of the lens, occupying one entire side of the vitreous cavity, the other half being filled with subretinal fluid. A necrotic focus of the tumor lay next to the sclera, the inner layers of which were necrotic, and the sclera was inflamed. Probably the patient suffered deep-seated pain from scleritis. In a third case there was failing vision for two months. Total detachment of the retina was seen two weeks before enucleation. For eight days the globe was inflamed and very painful, and the conjunctiva was chemotic. A hemispherical sarcoma sprang from the equatorial region of the choroid and extended to the median line of the globe. The retina remained adherent over the greater portion of the tumor, which was almost totally necrotic. The necrosis of the uveal tract was insignificant, but there was marked scleritis adjoining the tumor. Perhaps the lamina vitrea and the degenerated membranous retina prevented a great amount of toxins from diffusing into the vitreous space and reaching the uveal tract.

The Ciliary Vessels.—In several instances reference was made to enlarged anterior ciliary vessels. The ciliary vessels in the slides from

these cases were found to be thickened. All told, there were eleven cases of markedly hypertrophied ciliary vessels, which were mostly on the side of the tumor. In nine instances they were not seen in the specimens, and in sixty-four instances they were normal. The thickened ciliary vessels were found in cases of large, necrotic tumors with episcleral membranes.

The Tension.—In consideration of the prevalence of broad peripheral anterior adhesions of the iris and the relatively few times that the nerve showed a correspondingly deep excavation, the recording of the tension was of particular interest. There was a record in twenty-five cases. The tension was raised in nineteen cases, in four of which it was very high. In three cases it was normal, and in three there was hypotony. Anatomically the nineteen cases of high tension did not exhibit marked excavation of the nerve head. In the four cases in which tension was very high the cribriform fascia was but slightly pressed back. It is likely that congested blood vessels in and posterior to the cribriform fascia, together with edema, were factors that prevented a greater recession of the lamina cribrosa in many of the globes. The globe that contained the deepest excavation was that of a man aged 67. Blindness had been noted in this eye for five months and pain for five days. The tension was recorded as +2. Microscopically hyphemia was found. A broad peripheral anterior synechia was present, and the cribriform fascia was recessed, so that its entire anterior surface was on a level below that of the external surface of the sclera. The surface of the excavation was free from tissue except for a thin glial mantle. It was such an excavation as is commonly seen in chronic simple glaucoma, whereas most of the excavations were filled with edematous necrotic nerve substance. In this case the tension had probably been elevated over a long period, so that the excavation and disappearance of the papillary tissue preceded the necrosis of the tumor. The tumor was large and spherical, with a flat extension that surrounded the nerve head. An abscess was present in the nerve posterior to the cribriform fascia, on the side of the main mass of the tumor.

Exophthalmos.—There was recorded one case of pronounced exophthalmos. The patient had complained of photopsia for one year. For three months there was a peculiar reflex from the pupil. Violent inflammation existed for nine days before enucleation. Anatomic examination showed a large tumor between the posterior pole and the equator on one side. No living cell could be discovered in it in serial sections. The sclera over which it rested was almost completely liquefied. The exophthalmos must have been caused by orbital cellulitis. Exophthalmos was mentioned in other cases, in several of which excessive edema of the conjunctiva was noted, exactly as in panophthalmitis. There probably were still more instances of exophthalmos, judging by the number of

times inflammation and membranes were seen on the external surface of the globe.

Operations in Cases of Necrosis.—Operations had been performed in nine cases for the relief of pain or increased tension. A neurotomy was done on two patients, one of whom was aged 24 and the other 26. Microscopic examination showed nothing unusual in the necrosis in these two cases. The patient in the third case was 24 years old; at the age of 16 a staphyloma of the cornea was excised. Three weeks before enucleation sarcomatous nodules were discovered on the external surface of the globe. Microscopically the entire globe was filled with dense membranes, in the midst of which were many islands of sarcoma cells. In the fourth and fifth cases an iridectomy was performed, shortly after which removal of the globe became necessary owing to a return of pain and high tension. In the sixth case trephination was performed, and the pain was more severe after the operation than before. These three cases showed, in addition to the necrosis, an unusual amount of blood in the vitreous space. The seventh case was the one previously described in which a paracentesis was performed which seemed to have produced acute necrosis with extensive hemorrhage. In the eighth case the lens was removed because of senile cataract. A few days after the operation, the eye became violently inflamed and extremely painful. The wound gaped. It was believed that a nonexpulsive subchoroidal hemorrage had taken place. Microscopically a mushroom-shaped sarcoma was found, showing an almost totally necrotic head. In the ninth case there was no history other than that an iridectomy had been performed. Under the microscope, in addition to a mushroom-shaped tumor with a very necrotic base, there was seen a subchoroidal hemorrhage that was enclosed externally by a membrane that bridged a defect in the sclera caused by a spontaneous rupture.

Injuries and Sympathetic Ophthalmitis.—There were two cases in which the histories spoke of inflammation in the fellow eye, causing a diagnosis of sympathetic ophthalmitis to be made. In the first case sympathetic ophthalmia appeared six months before enucleation. Microscopic examination showed that at the limbus there was an old retracted scar continuous with a cyclitic membrane that surrounded the lens. There was a large, almost completely necrotic sarcoma of the choroid. The choroid on the side opposite the tumor showed many typical foci of sympathetic ophthalmia. It is probable that the sympathetic inflammation was due to injury rather than to necrosis of the sarcoma. The second case was that of a man, aged 42, in whom detachment of the retina was discovered fifteen months before enucleation. For two months he suffered great pain, and shortly before enucleation severe iridocyclitis developed in the fellow eye. Anatomically an infiltrating sarcoma, partially necrotic, replaced half of the choroid. The other half showed typical sympathetic nodules. It is noteworthy that of eighty-

four choroidal sarcomas that had passed through every stage in the life history of an intra-ocular sarcoma, sympathetic ophthalmia occurred in only one indisputable case.

COMMENT

The anatomic survey demonstrated that no tissue within the globe can withstand the death-dealing toxins generated in a necrotic sarcoma. The toxins by diffusing through the sclera or by destroying it may set up an active inflammation in the orbital tissues. The clinical signs and symptoms are explained by the pathologic changes. The chocolate-colored content of the anterior chamber is an indication of acute necrosis, whereby with the rupture of engorged blood vessels and the bursting of swollen pigment cells, blood and pigment granules escape and become mixed in the aqueous. Chemosis of the conjunctiva and edema of the lids, together with exophthalmos, are expressions of orbital cellulitis. To the pain and vomiting of glaucoma may be added the pain of iridocyclitis and scleritis. These symptoms, suddenly appearing in an eye that gives a history of failing vision, especially when the fellow eye is free from glaucoma, are so characteristic of necrosis in sarcoma as an entity that with careful examination, including verification with transillumination, a correct diagnosis should be made. Indeed, a forecast of impending general necrosis in a sarcoma may be made with the ophthalmoscope if one observes that on the surface of the tumor, which has hitherto been of a uniform grayish color, there appear pigmented spots. Such dark areas may indicate necrosis because the cells of a sarcoma contain a prepigment substance that has the property of changing into pigment under the influence of toxins released in the process of necroses. Localized pain elicited on palpation over the sclera is suggestive of necrosis in the external layers of a sarcoma.

If, however, a tumor is not suspected and an operation for the relief of pain and high tension is performed, the opening of the globe may have its laudable aspect. The rapid diminution of pressure may cause ruptures in the thin-walled vessels of the tumor and with the extravasation of blood induce acute necrosis, as seemed to have happened in several of our cases; as a consequence the symptoms soon became worse than they were before, necessitating enucleation without further delay.

According to the anatomic division of the survey, the twenty cases of totally necrotic tumors represent a spontaneous local cure in 23.8 per cent of the eighty-four cases. Fifteen tumors, or 16.6 per cent, had extensions in the orbital tissues. There was no indication in the clinical records that metastasis was ever discovered at the time of enucleation. It is astonishing to realize for how long a time a sarcoma of the uvea may show local manifestations and the degenerative changes that it may undergo without clinical evidence of transplantation in remote parts of the body.

REPAIR OF COLOBOMA OF THE UPPER EYELID

LYNDON ARTHUR PEER, M.D.
NEWARK, N. J.

In the case reported here coloboma followed the removal of a portion of the upper eyelid for basal cell carcinoma. The growth involved both the conjunctiva and the skin and was removed by excising a full thickness section of the lid extending from the free margin to a little above the upper border of the tarsus. Following operation, a small portion of the eyelid near the inner canthus remained, with the median palpebral ligament. Five months later there was no sign of recurrence, but the conjunctiva was becoming increasingly irritated from exposure. For repair of this defect, it is essential to have a lining to replace the conjunctiva and a cutaneous surface for the outside of the lid. In this case the remaining fibers of the orbicularis oculi muscle above and at the sides of the defect provided good closures, and sufficient of the levator attachment remained to elevate the lid. A cartilage support to replace the tarsus is not necessary, providing adhesions are made between the upper and the lower eyelid to prevent vertical contracture. Mucous membrane is a better substitute for conjunctiva when a central coloboma is present exposing the cornea, as skin will irritate the cornea, but when the coloboma is located near the inner or the outer canthus, cutaneous lining may be used, as horizontal contracture of the graft tends to pull the normal border of the lid over the cornea.

The operation described in this article utilizes the skin of the adjacent lid for a lining and a free, full thickness skin graft from the opposite upper eyelid for a covering. It is best used when the loss does not extend far above the upper border of the tarsus and is not centrally located.

PROCEDURE

A flap of skin from the eyelid was outlined above the defect, dissected from the muscle layer and turned down trapdoor fashion, sufficient hinge or pedicle being left to insure an adequate supply of blood. This flap was sutured to the denuded edges of the defect with the surface of the skin next to the eye and the raw surface outside, and was left in place for one week. A free, full thickness skin graft was then taken from the opposite upper eyelid and sutured to cover the entire denuded area, only moderate pressure being applied to avoid strangulation of the skin flap. One week later the dressing was removed, and artificial adhe-

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From the Plastic Surgery Department of the Newark Eye and Ear Infirmary.
Presented at the clinical meeting of the Academy of Medicine of Northern New Jersey, Nov. 13, 1933.

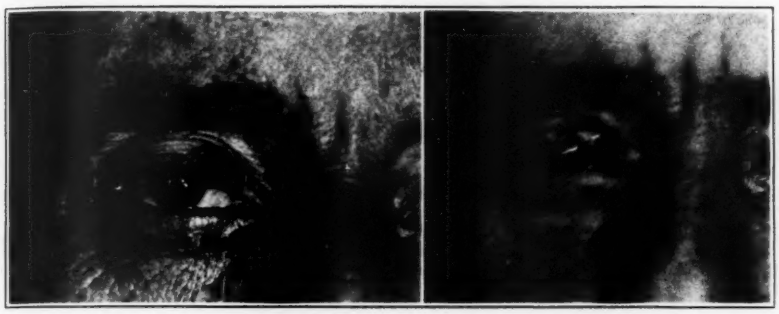

Fig. 1.—*A*, basal cell carcinoma of the right upper eyelid involving both the conjunctiva and the skin. *B*, the defect resulting five months after excision of the growth.

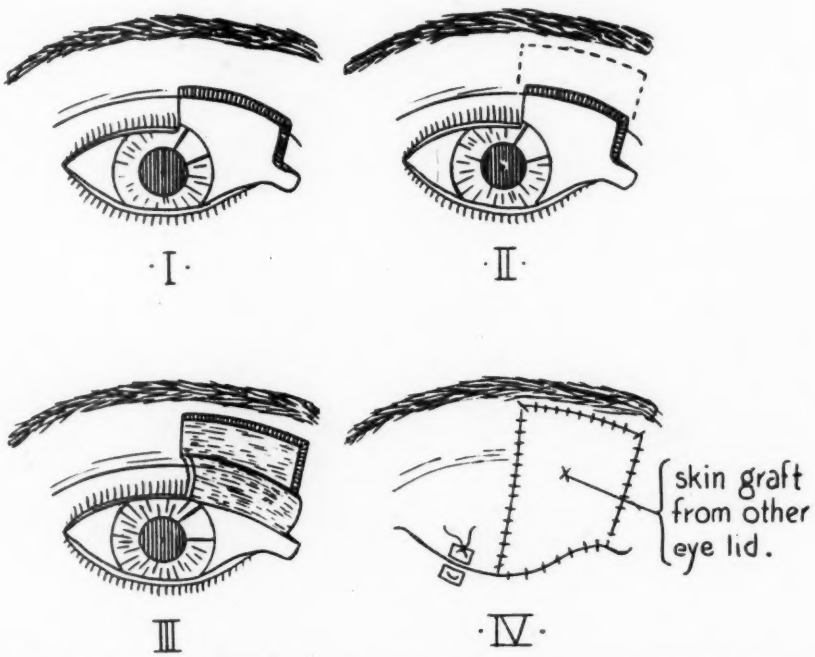

Fig. 2.—Diagrams illustrating the method of repair: *I*, defect in the eyelid; *II*, trapdoor of skin outlined; *III*, skin dissected from the muscle and turned down to fill the defect; *IV*, free, full thickness skin graft from the opposite upper eyelid to cover the denuded area.

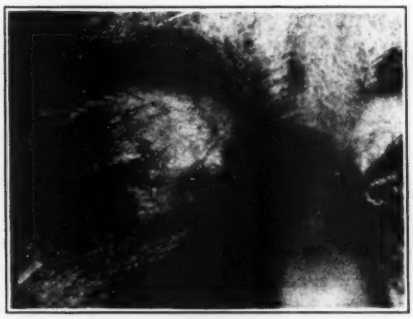

Fig. 3.—Result three months after operation. Adhesions hold the upper and the lower eyelid together. A notching of the upper lid is present, owing to imperfect union.

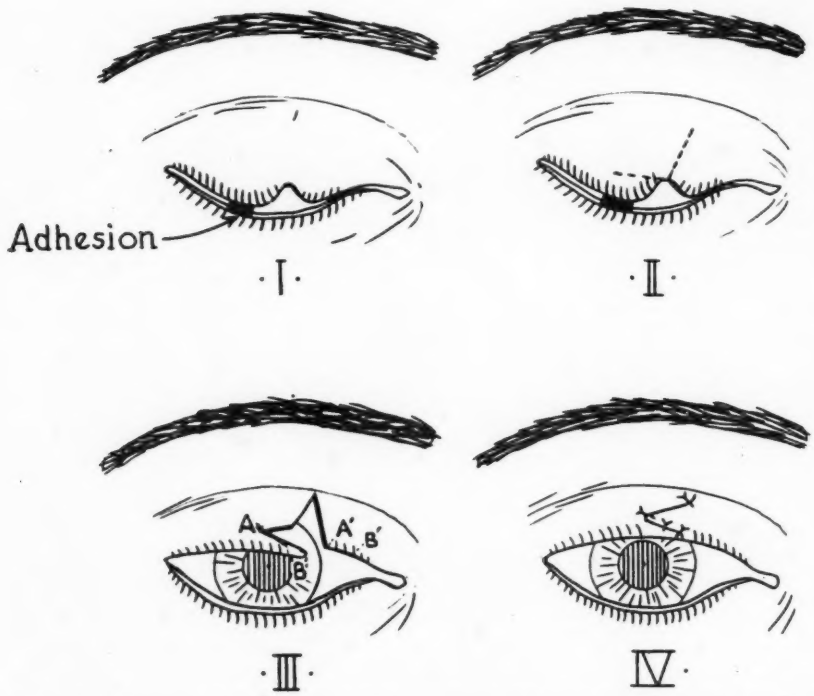

Fig. 4.—Diagrams illustrating repair of the notch: *I*, notch in the upper eyelid with artificial adhesions binding the lids together; *II*, two incisions (dotted lines) made through the full thickness of the eyelid. *III*, the margin of the lids is denuded between *A'* and *B'*, a distance corresponding to *AB*, as shown in *III*; *B* is sutured to *B'*, and *A* is sutured to *A'*, lengthening the lid and obliterating the notch, as shown in *IV*.

sions were made between the new portion of the upper and lower lids. The adhesions were left in place four months, and then severed. A notching of the margin of the lid due to imperfect union was repaired at this time by a procedure similar to the Blair-Mirault[1] operation for harelip.

I have used this operation on other cleft lids and can recommend it as a practical and satisfactory method. The patient shown here obtained an excellent functional and cosmetic closure. The palpebral fissure is

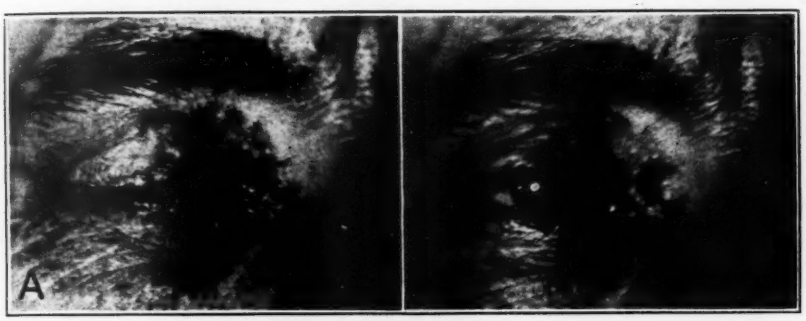

Fig. 5.—*A,* final result nine months after operation with lids closed. The approximation is accurate. *B,* final result with the lids open. Note the normal width of the palpebral fissure.

normal in width with the lid raised, and the margins meet accurately when the lids are approximated. The congestion of the conjunctiva present before operation disappeared almost completely, but did not entirely subside until the patient was instructed to use a small amount of corrosive mercuric chloride ointment under the lower lid twice a day. When last seen nine months following his operation, the cornea and conjunctiva were normal, and the vision was 20/20 in each eye.

1. Blair, V. P., and Brown, J. B.: Mirault Operation for Single Harelip, Surg., Gynec. & Obst. **51**:81 (July) 1930.

Ophthalmologic Review

EDITED BY DR. FRANCIS HEED ADLER

ANEURYSMS OF THE CIRCLE OF WILLIS

PAUL H. GARVEY, M.D.

ROCHESTER, N. Y.

During the past decade the clinical syndrome produced by aneurysms at the base of the brain has been the subject of several papers, with special reference to diagnosis during life. In a number of cases included in these reports a definite diagnosis of intracranial aneurysm was established during life and was subsequently verified at operation or necropsy. The earlier writers on the subject described rather uniform clinical symptoms as a result of their analyses of large series of cases, yet they felt that the clinical diagnosis was very uncertain.

In 1859, Sir William Gull [1] called attention to the relative frequency of intracranial aneurysms and their clinical manifestations. After a survey of previously reported cases and 7 of his own, he concluded: "Although we may from the circumstances sometimes suspect the presence of an aneurysm within the cranium, we have at the best no symptoms on which to ground more than a probable diagnosis." Beadles,[2] writing over fifty years later, arrived at the same conclusion after analyzing a series of 555 cases of intracranial aneurysms. He stated: "Only two or three have ever been diagnosed during life, and even in these cases it can scarcely be said to have been an absolutely certain diagnosis."

Fearnsides,[3] in 1916, gave an excellent review of the subject. He maintained that the original statement of Gull still held true, although in those cases in which multiple leakage of blood occurred owing to partial rupture of the aneurysmal sac, the clinical manifestations formed a definite nosologic group, rendering their diagnosis not difficult. He attributed the infrequency of diagnosis during life to the fact that the possibility of aneurysm was rarely considered.

From the Department of Medicine, the School of Medicine and Dentistry, University of Rochester and the Medical Clinic of the Strong Memorial and Rochester Municipal Hospitals.

1. Gull, W.: Cases of Aneurysm of the Cerebral Vessels, Guy's Hosp. Rep. **5**:281, 1859.

2. Beadles, C. F.: Aneurysms of the Larger Cerebral Arteries, Brain **30**:285, 1907.

3. Fearnsides, E. G.: Intracranial Aneurysms, Brain **39**:224, 1916.

In 1923, Symonds [4] reported 5 cases of intracranial aneurysm in which the diagnosis was made during life. In 2 of his cases the diagnosis was confirmed by necropsy; in another case the necropsy was incomplete, and in the remaining 2 the patients were still living. This report served as the turning point from the time-honored tradition that an exact diagnosis of intracranial aneurysm was impossible during life. During the past few years several other papers have appeared containing the reports of cases in which a definite clinical diagnosis was established. Albright [5] reported 2 of his own cases in which a diagnosis was made before death. In 1 case the diagnosis was verified at necropsy. Pfingst and Spurling [6] described the clinical manifestations in 2 unverified cases of intracranial aneurysm and emphasized the rôle played by intracranial aneurysm in the production of ocular palsy. Lemmel [7] reported 3 cases in which a clinical diagnosis of intracranial aneurysm was made before death and was confirmed at necropsy.

My object in this paper is to review briefly the clinical and pathologic features in aneurysmal dilatations of macroscopic size involving the vessels of the circle of Willis, with special emphasis on the clinical symptoms and signs which lead to their recognition during life. The clinical findings and pathologic changes in 4 cases of intracranial aneurysm and the clinical findings in a fifth case are reported. In cases 1 and 2 a diagnosis of intracranial aneurysm was made and later verified at necropsy. In cases 3 and 4 the clinical syndrome was ascribed to meningovascular syphilis and cerebral tumor, respectively. The fifth patient is still living, but the diagnosis of aneurysm seems almost certain.

REPORT OF CASES

CASE 1.—*Pain in the left supra-orbital region two months before admission. Sudden onset of severe generalized headache and oculomotor paralysis with subarachnoid bleeding. Transient right hemiplegia and aphasia. Sudden death.*

History.—I. L., a housewife, aged 43, was admitted to the Rochester Municipal Hospital on July 20, 1932. She complained of severe fronto-occipital headache and inability to open the left eye. Her present illness began two months prior to admission with sudden, severe pain localized over the left eye. The pain continued until ten days before the patient's admission, when she was seized with a sudden, severe, generalized headache, vomiting, numbness of the right leg and drooping

4. Symonds, C. P.: Contributions to the Clinical Study of Intracranial Aneurysms, Guy's Hosp. Rep. **73**:139, 1923.

5. Albright, F.: The Syndrome Produced at or Near the Junction of the Internal Carotid Artery and the Circle of Willis, Bull. Johns Hopkins Hosp. **44**: 215, 1929.

6. Pfingst, A. O., and Spurling, R. G.: Intracranial Aneurysms: Their Rôle in Production of Ocular Palsies, Arch. Ophth. **2**:391 (Oct.) 1929.

7. Lemmel, G.: Drei klinisch diagnostizierte Fälle von Aneurysmen der Hirngefässe, München. med. Wchnschr. **79**:2193, 1931.

of the left eyelid. The onset of these symptoms was accompanied by a sensation which, according to the patient, was "as if all my blood rushed to my head." The headache persisted, and the drooping of the eyelid increased until the day of admission.

Previous to her present illness, the patient enjoyed good health. Three years before admission to the hospital she was told that she had mild Bright's disease. Hypertension was discovered a few days prior to admission. The family history was irrelevant, except that the mother died of cerebral hemorrhage.

Examination.—On admission, the patient was acutely ill and in great discomfort, owing to intense headache. She was mentally clear. The temperature was 38 C. (100.4 F.); the pulse rate, 84, and the respiratory rate, 25. There was complete ptosis of the left eyelid. The left eyeball was turned upward and outward, and the only movement possible was lateral rotation. The left pupil was widely dilated and immobile. Ophthalmoscopic examination gave negative results. The visual fields were normal. The oculomotor nerve was the only cranial nerve involved. Neurologic survey of the extremities gave negative results except for a bilateral Kernig sign. The neck was stiff. A bruit could not be heard over the cranium.

General physical examination gave negative results, except for slight evidence of hypertensive heart disease. The peripheral vessels showed no evidence of sclerosis. The blood pressure was 190 systolic and 105 diastolic.

Lumbar puncture revealed a uniformly bloody spinal fluid under a pressure of 250 mm. of water, with 90,000 red and 200 white blood cells per cubic millimeter. On centrifugation of the spinal fluid, the supernatant fluid was xanthochromic. The Wassermann tests of the blood and spinal fluid were negative. A blood count showed 80 per cent hemoglobin, 5,000,000 red cells and 15,000 white cells per cubic millimeter. The urine showed a trace of albumin. Chemical examination of the blood gave negative results.

Clinical Notes.—July 25: The patient complained of pain over the left eye. The neck was less stiff. Lumbar puncture showed a xanthochromic spinal fluid under a pressure of 148 mm. of water and containing 700 red blood cells per cubic millimeter.

July 26: During the evening, right hemiplegia and motor aphasia suddenly developed.

July 27: The muscle power returned to the right extremities.

August 2: Lumbar puncture revealed a clear, colorless fluid. The spinal fluid pressure was 125 mm.

August 4: At 5:15 a. m. the patient was found unconscious, frothing at the mouth and gasping for breath. The blood pressure at this time was 190 systolic and 100 diastolic. She died one-half hour later from respiratory paralysis. A lumbar puncture done after the respirations stopped showed a grossly bloody fluid under a pressure of 280 mm. of water.

Clinical Diagnosis.—The condition was diagnosed as intracranial aneurysm with rupture.

Necropsy.—Gross Anatomy: The postmortem examination was limited to the brain. On opening the dura, considerable blood escaped. The base of the brain about the circle of Willis showed considerable blood clot adherent to the pia-arachnoid. The vessels of the circle of Willis were thin and elastic. About 4 mm. from the point of origin of the left middle cerebral artery, the wall of the vessel was thinned out, and there was a bulging aneurysm which measured 4 by 5 mm., which had ruptured (fig. 1). The inner surface of the aneurysm was covered with

a thin layer of friable blood clot. On section of the brain nothing unusual was noted, except that the ventricles were filled with blood.

Microscopic Examination: Histologic study of the wall of the aneurysm was unsatisfactory.

Anatomic Diagnosis.—A diagnosis of aneurysm of the left middle cerebral artery (presumably due to arteriosclerosis) with rupture was made.

Comment.—The diagnosis of aneurysm of the anterior portion of the circle of Willis seemed almost certain, in view of the oculomotor paralysis and evidence of recurrent subarachnoid bleeding. Apparently the first leakage from the aneurysm occurred on July 10 with the onset

Fig. 1 (case 1).—Ruptured aneurysm of the left middle cerebral artery.

of severe generalized headache, vomiting and the gradual development of palsy of the third nerve. The pain localized in the right supra-orbital region, which existed for two months prior to admission, was probably due to irritation of the ophthalmic division of the fifth nerve. Transient hemiplegia has been regarded as a common symptom in aneurysms arising from both the anterior and the posterior portion of the circle of Willis.

The history of symptoms indicative of subarachnoid hemorrhage is more important in determining whether leakage has occurred than the character of the spinal fluid at a single examination, unless the puncture is done at the height of the attack or within a few days thereafter. The exact rate of absorption of blood from the subarachnoid space, to my

knowledge, has never been definitely determined. In this case a cherry-red spinal fluid was obtained on July 20 containing 90,000 red blood cells per cubic millimeter, and two weeks later the spinal fluid was entirely normal.

CASE 2.—*Chronic headache for several years with an acute exacerbation one week before admission followed by convulsions, proptosis of the right eye and paralysis of the right oculomotor nerve. Sudden death.*

History.—C. R., aged 54, married, a shoemaker, was admitted to the Strong Memorial Hospital, on Feb. 3, 1933, in a semicomatose condition. His wife said that he had complained of chronic headaches for several years, but that for the past four weeks they had been more severe. On the morning of January 26, the patient was awakened by a severe headache and pain in the neck and spine. The onset of this excruciating headache was followed by loss of consciousness and a series of four tonic convulsions. The patient was removed to a hospital, and a spinal puncture yielded a bloody spinal fluid. Four days previously, ptosis of the right eyelid, proptosis of the right eye and weakness of the facial muscles on the lower left side developed. The past history was irrelevant.

Examination.—On admission, the patient was semicomatose. The temperature was 39 C. (102.2 F.); the pulse rate, 100, and the respiratory rate, 35. Ptosis of the right eyelid and nonpulsating exophthalmos on the right were noted. The pupils were unequal. The right pupil was dilated and immobile, and the left responded promptly to light. The extra-ocular movements were difficult to test, but the limitation of movement of the right eye indicated paralysis of the oculomotor nerve. Ophthalmoscopic examination revealed numerous retinal hemorrhages in the right eye. A large subhyaloid hemorrhage surrounded the inferior margin of the right disk. The retinal vessels showed moderate sclerosis. A bruit could not be heard over the cranium. The tendon reflexes on the left were increased. There was moderate stiffness of the neck.

General physical examination gave negative results, except for slight cardiac enlargement and some evidence of pneumonia at the base of the left lung. The blood pressure was 124 systolic and 74 diastolic. The roentgenograms of the skull were negative.

Lumbar puncture revealed a uniformly bloody spinal fluid under a pressure of 400 mm. of water. A blood count showed 12,500 white cells per cubic millimeter. The Wassermann test of the blood was negative.

Course.—The patient's condition remained about the same until his sudden death three days following admission. On the morning of February 6 he had a choking spell; Cheyne-Stokes respirations developed and death occurred shortly thereafter.

Clinical Diagnosis.—A diagnosis of intracranial aneurysm was made.

Necropsy.—Gross Anatomy: The observations of interest were limited to the intracranial structures. On removing the calvarium, free blood was present in the subdural space. The base of the brain was covered with a soft, jelly-like clot, and when the clot was washed away a grayish, much firmer, friable clot was found in the region of the circle of Willis. At the point of division of the right internal carotid artery into the middle and anterior cerebral arteries was a small, delicate, thin-walled aneurysm, which had ruptured (fig. 2). The undersurface of the right temporal lobe was torn and discolored and contained a large blood clot. On section, the third and fourth ventricles were filled with a blood clot. In the

temporal horn of the right lateral ventricle adjacent to the area of destruction, there was a small, firm blood clot.

Microscopic Examination: The aneurysmal sac showed no inflammatory reaction. The media was entirely absent in some portions. Sections of the other vessels at the base of the brain showed marked hyaline thickening of the intima.

Anatomic Diagnosis: The diagnosis was aneurysm of the right internal carotid artery with rupture into the subarachnoid and subdural spaces and the temporal lobe and generalized arteriosclerosis.

Comment.—The history of recurrent attacks of subarachnoid bleeding followed by palsy of the third nerve and subhyaloid hemorrhage

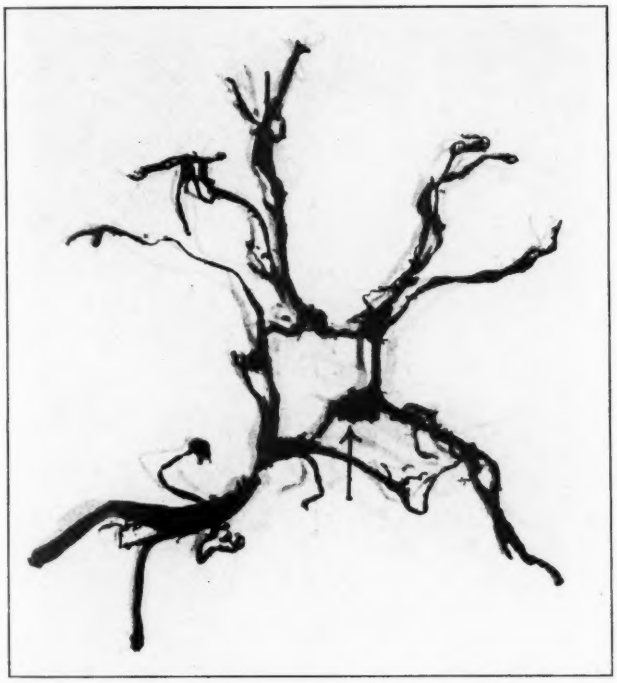

Fig. 2 (case 2).—Ruptured aneurysm of the right internal carotid artery.

made the diagnosis of aneurysm almost certain. Operative treatment (ligation of the right internal carotid artery) was considered, but unfortunately it was deferred on account of the pulmonary complication. The patient's sudden death a few days later was apparently due to a sudden increase in intracranial pressure resulting from a paroxysm of choking. It is perhaps doubtful if ligation of the internal carotid artery would have prevented the fatal rupture. However, in view of the fatal outcome of such a high percentage of these cases without treatment, it would be justifiable to ligate the artery in cases in which the diagnosis is reasonably certain. The operation has not been done in a sufficient number of cases to warrant definite conclusions regarding its value as a therapeutic measure.

CASE 3.—*Drooping of the right eyelid, double vision and pain in the right side of the face following an attack of loss of consciousness two and one-half years prior to admission. Changes in the blood and the spinal fluid. Death due to bronchopneumonia. Unruptured aneurysm of the right internal carotid artery.*

History.—E. D., aged 55, a housewife, was admitted to the emergency division of the Strong Memorial Hospital on April 2, 1932. She complained of drooping of the right eyelid, double vision and constant pain in the right side of the face. These symptoms followed a sudden attack of loss of consciousness two and one-half years previously. Other symptoms of more recent development were weakness of the legs, urinary incontinence, difficulty in swallowing, dizziness and failing vision.

Fig. 3 (case 4).—Large unruptured aneurysm of the right internal carotid artery.

Three years prior to admission she was in rather poor health. A pelvic operation at this time failed to relieve her. In 1930, a positive Wassermann reaction of the blood was discovered, and she received a series of twenty-six intramuscular injections of antisyphilitic therapy.

Examination.—The patient did not appear acutely ill. The temperature, pulse and respirations were normal. She was mentally clear, but her memory for recent events was poor. There was complete ptosis of the right eyelid. The right eye deviated upward and outward, and the only movement which could be performed

was external rotation. The position and movements of the left eye were normal. The right pupil was dilated and fixed; the pupillary reactions of the left eye were sluggish. Ophthalmoscopic examination revealed a bilateral chorioretinitis and primary optic atrophy in the right eye. The other cranial nerves were normal except for a peripheral type of weakness of the seventh nerve on the right side. Neurologic survey of the extremities revealed a spastic weakness of the lower extremities with increased tendon reflexes and a bilateral Babinski sign. Deep sensibility was impaired in the legs.

The results of the general physical examination were negative. The blood pressure was 135 systolic and 85 diastolic. A spinal puncture yielded a clear colorless fluid under a pressure of 300 mm. of water. The Pandy test was positive. A cell count showed 80 red blood cells per cubic millimeter. The Wassermann test of the blood with the cholesterolized antigen was 2 plus and of the spinal fluid, 4 plus. The colloidal gold curve was 1122321000.

The patient was discharged from the hospital on April 9, with a diagnosis of meningovascular syphilis. She was given a course of eight injections of thiobismol, which was followed by a course of thirteen injections of bismarsen without improvement in her condition. On Jan. 9, 1933, she was admitted to the hospital with signs and symptoms of bronchopneumonia, and died the following day from cardiac failure.

Clinical Diagnosis.—A diagnosis of meningovascular syphilis and bronchopneumonia was made.

Necropsy.—Gross Anatomy: The examination of the brain revealed a round tumor, 3 cm. in diameter, situated in the interpeduncular space (fig. 3). It compressed the right optic tract, right temporal lobe, right peduncle, midbrain and pons. On section, it had the characteristic appearance of an aneurysm which apparently involved the right internal carotid artery. There was no evidence of rupture. In the first portion of the right middle cerebral artery there was an organizing thrombus. The other basilar vessels showed no abnormality except moderate thickening. Section of the brain showed no evidence of hemorrhage or softening.

Microscopic Examination: The wall of the aneurysm was composed of thick fibrous tissue, which in some places was infiltrated with lymphocytes and plasma cells. The histologic study of other vessels comprising the circle of Willis and its branches failed to show changes which might be regarded as syphilitic arteritis. There was moderate intimal thickening. The examination of other organs showed marked arteriosclerosis but no changes in the vessels characteristic of syphilis.

Anatomic Diagnosis.—A diagnosis of generalized arteriosclerosis and aneurysm of the right internal carotid artery was made.

Comment.—The presence of an intracranial aneurysm was not suspected in this case. The clinical picture seemed to be adequately explained by a syphilitic basilar meningitis. One of the common and characteristic lesions of syphilis of the nervous system is a meningitic process at the base which results in paralysis of one or more cranial nerves. The oculomotor and the optic nerves are frequently affected. This case also demonstrates the difficulty in making the diagnosis of aneurysm before rupture occurs.

CASE 4.—*Sharp paroxysmal pain in the right side of the face; drooping of the right eyelid of nine months' duration. Progressive ophthalmoplegia and proptosis of the right eye. Section of the sensory root at the fifth nerve for relief from pain. Death.*

History.—A. C., aged 64, a housewife, was first admitted to the gynecologic service of the Rochester Municipal Hospital on Aug. 14, 1929, for operative treatment of a urethral caruncle and cervical polyp. Her general health at the time was excellent. The blood pressure was 170 systolic and 80 diastolic. The Wassermann test of the blood was negative. Complete relief from the local symptoms followed the operation.

The patient was not seen again until May 9, 1932, when she came to the medical clinic with the complaint of drooping of the right eyelid and attacks of pain in the right side of the face. During the summer of 1929, her friends noticed that she was "cross-eyed." In December, 1929, she first noticed drooping of the eyelid, and two months later attacks of severe pain in the right temporal region. The ptosis of the lid gradually increased during the six months' period prior to examination. There was no history of headache, diplopia or loss of consciousness. The past history was unimportant. Hypertension was discovered three years previously but was asymptomatic.

Examination.—The patient was an elderly, obese woman, not acutely ill. She was mentally clear. Partial ptosis of the right eyelid was associated with intermittent spasm of the orbicularis oculi muscles. The right pupil was dilated and fixed. The pupillary reactions and the extra-ocular movements of the left eye were normal. On the right side there were complete paralysis of the external rectus muscle and limitation of upward and downward movement. The function of the internal rectus muscle was normal. The ocular fundi were normal except for a moderate degree of retinal arteriosclerosis. There was no gross defect of the visual fields. The left upper trigeminal field showed slight hyperesthesia and hyperalgesia. A bruit was not audible over the skull. The positive neurologic signs were limited to the right oculomotor, trigeminal and abducens nerves. General physical examination showed some cardiac enlargement. The blood pressure was 180 systolic and 85 diastolic. There was no evidence of a tumor in the nasopharynx.

Stereoscopic roentgenograms of the skull showed an extensive area of hyperostosis over the frontal bone and erosion of both the anterior and the posterior clinoid processes. A small area of calcification was seen behind the posterior clinoid processes.

A diagnosis of tumor of the middle fossa was made, and admission to the hospital and operation were advised. The patient declined to follow this advice.

On Jan. 27, 1933, she returned to the hospital because of excruciating pain over the right side of the face and projectile vomiting of five days' duration. Examination revealed complete paralysis of the third, fourth and sixth nerves on the right side. There was slight exophthalmos on the right. Pain and tactile sensations were diminished over the right upper trigeminal field. The muscles of mastication were weak on the right side. The fundi were normal except for retinal arteriosclerosis. Neurologic survey of the extremities revealed no abnormalities. The heart was enlarged, but there was no evidence of congestive failure. The blood pressure was 200 systolic and 100 diastolic.

The problem at this stage seemed to be the selection of some method of relieving the trigeminal pain. After considerable deliberation, it was decided to section the posterior root of the fifth nerve by the cerebellar route.

Operation (Dr. W. P. Van Wagenen).—January 30: With the patient under ether anesthesia, the dura over the cerebellum was exposed. It appeared very blue, and on incision a blood clot was found in the subdural space. The blood clot covered the superior and the lateral surface of the cerebellum and extended into the cerebellopontile angle. The clot was removed with the aid of suction and the fifth nerve was exposed and divided.

After recovering from the anesthesia, the patient gradually lapsed into a semicomatose state from which she did not recover. Repeated lumbar punctures following operation showed a bloody spinal fluid. Death occurred on February 7.

Clinical Diagnosis.—The condition was diagnosed as cerebral tumor.

Necropsy.—Gross Anatomy: The cerebral portion of the right internal carotid artery was represented by a large mass measuring 4 by 6 cm., which was purplish and firmly attached to the underlying dura. The aneurysmal mass showed evidence of rupture on its posterior surface, and a laminated grayish-white clot exuded from the point of rupture. There was an indentation of the undersurface of the right frontal lobe corresponding to the position of the aneurysm. The aneurysm also eroded the lateral portion of the sella turcica and compressed the left optic nerve proximal to the chiasm. In addition to the large aneurysm there were several small oval-shaped outpouchings at the points of bifurcation of the smaller branches of the middle cerebral artery.

Microscopic Examination: The wall of the large aneurysm was made up of thickened, hyalinized intima, thin media and dense fibrous connective tissue in which a ganglion cell was seen. Other basilar vessels showed marked intimal thickening.

Anatomic Diagnosis.—A diagnosis of aneurysm of the right internal carotid artery with rupture was made.

Comment.—The mode of development of the syndrome presented in this case was more characteristic of a neoplasm than of an aneurysm. During the preoperative course the diagnosis of an aneurysm was not considered, owing chiefly to the absence of signs and symptoms of leakage from the sac. The differential diagnosis between neoplasm and aneurysm in cases of this type rests on the correct interpretation of the roentgenograms. Sosman [8] described rather characteristic changes in the roentgenograms in patients with aneurysms of the internal carotid artery. They may show: (1) erosion of a clinoid process or of a lateral half of the sella, or (2) calcification in the wall of the aneurysm or in the laminated clot in its sac.

CASE 5.—*Onset six weeks before admission with a generalized convulsion followed by headache and vomiting; recurrent headache and paralysis of the left oculomotor nerve; transient hemiplegia. Partial recovery.*

History.—A. F., aged 40, a housewife, was admitted to the Strong Memorial Hospital on Aug. 1, 1932. She complained of pain over the right eye. Six weeks before admission she suddenly fainted and fell to the floor. A few minutes later she had a tonic convulsion, which was followed by severe headache and vomiting. She remained in a semicomatose condition for three days. During the course of the next three weeks her symptoms gradually disappeared, so that she was able

8. Sosman, M. C.: Aneurisms of the Internal Carotid Artery and Tumors of the Base of the Brain, Brit. J. Radiol. **30**:468, 1925.

to leave her bed. Three days before admission she was awakened with severe occipital headache and was surprised to find that she was unable to open the right eye.

The past history was of little interest except that the patient had been under observation and had received treatments for pelvic inflammatory disease in the outpatient department for a period of three years, during which time she had no symptoms referable to the head or cranial nerves.

Examination.—The patient appeared acutely ill. She was mentally clear. The temperature was 37.5 C. (99.5 F.); the pulse rate, 90, and the respiratory rate, 20. There was complete ptosis of the right eyelid. The right eye was turned upward and outward, and the only movement possible was external rotation. The right pupil was dilated and immobile. Movements of the left eye were normal. The left pupil responded promptly to light and in accommodation. Ophthalmoscopic examination gave negative results. The other cranial nerves were normal. A bruit could not be heard over the cranium. Neurologic survey of the extremities revealed no abnormalities. There were some resistance and pain associated with passive flexion of the head but no other signs of meningeal irritation.

The results of the general physical examination were negative. The blood pressure was 115 systolic and 70 diastolic.

Lumbar puncture yielded a bloody spinal fluid under a pressure of 100 mm. of water with 360,000 red blood and 480 white blood cells per cubic millimeter. The Pandy test was strongly positive. A blood count showed 85 per cent hemoglobin, 4,240,000 red cells and 12,000 white cells per cubic millimeter. The Wassermann tests of the blood and spinal fluid were negative. Urinalysis gave negative results.

Clinical Notes.—The patient's condition improved gradually with rest in bed and repeated lumbar punctures. On August 12 she was free from headache, although the spinal fluid was still xanthochromic and contained about 10,000 red blood cells per cubic millimeter. On the evening of this day she suddenly lost consciousness and her condition became precarious. During the next few days she remained in a semicomatose condition. On August 17 left hemiparesis developed, which progressed to complete paralysis two days later. Ten days after the onset of the hemiplegia, she gradually regained strength in the left extremities and at the time of discharge from the hospital, September 18, the only residual findings were complete paralysis of the right oculomotor nerve and increased tendon reflexes in the left extremities.

Since her discharge from the hospital she has been examined on several occasions. She has had no recurrence of the subarachnoid bleeding. At the time of her last examination, Jan. 24, 1933, there was some evidence of improvement of the oculomotor paralysis.

Clinical Diagnosis.—The condition was diagnosed as aneurysm of the anterior portion of the circle of Willis.

Comment.—The similarity of the history and clinical findings to those in case 1 and 2 is rather striking. While the confirmatory evidence is lacking, the clinical course has been sufficiently characteristic to warrant the diagnosis of intracranial aneurysm.

INCIDENCE

Sir William Gull was the first writer to call attention to the relative frequency of aneurysms of the basilar vessels. He attributed the appa-

rent infrequency previous to that time to careless inquiry, and he further emphasized that unless a minute dissection is made the aneurysmal sac may not be discovered. The incidence of intracranial aneurysm reported by more recent authors leads one to believe that it is not a rare disease. Schmidt [9] stated that genuine aneurysms of the type under discussion appear in about 0.5 to 1 or 1.5 per cent of all postmortem examinations, though sometimes the frequency is stated to be less. The 4 verified cases reported in this paper are the total number of cases encountered in approximately 200 autopsies done over a period of seven months.

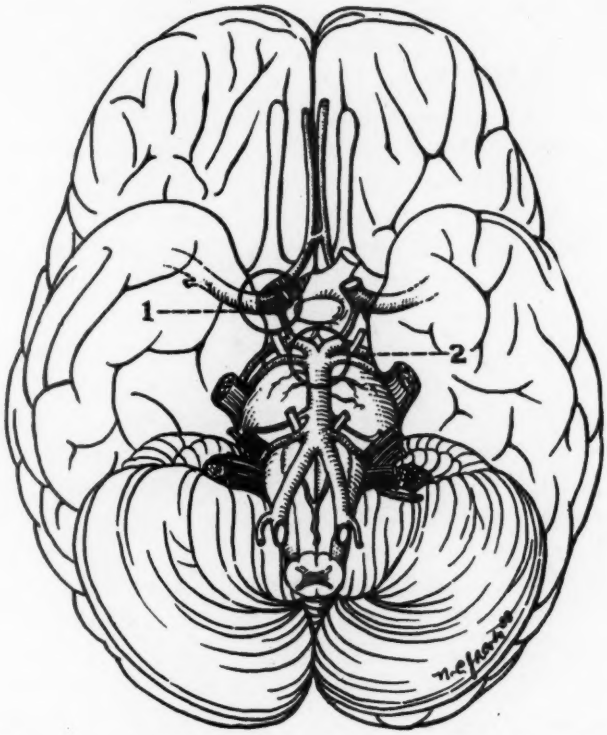

Fig. 4.—Drawing of the base of the brain and the circle of Willis. The circles indicate two common sites for aneurysmal formation.

The relative frequency with which different vessels at the base of the brain are involved varies somewhat, depending on the number of cases in the series. Gowers [10] reported the following distribution in a series of 154 cases studied: middle cerebral, 44; basilar, 41; internal carotid, 23; anterior cerebral, 14; posterior communicating, 8; anterior communicating, 8; vertebral, 7; posterior cerebral, 6; inferior cerebellar, 3. The statistical studies which have been published since then indicate

9. Schmidt, M.: Intracranial Aneurysms, Brain 53:489, 1931.

10. Gowers, W. R.: A Manual of Diseases of the Nervous System, London, J. & A. Churchill, 1888, vol. 2, p. 496.

that the internal carotid artery is affected more frequently than the basilar artery. In general, it might be said that aneurysms of the carotid portion of the circle of Willis are far more common than aneurysms of the vertebral system (fig. 4). They occur usually at the point of junction of two arteries or at the origin of a branch. The middle cerebral vessels are affected more frequently than any other pair of vessels. Aneurysms of the cerebellar vessels are extremely rare.

It has been frequently asserted that intracranial aneurysms are more common on the left side, owing to the fact that the carotid artery arises directly from the aorta. According to Gowers, aneurysms are rather more common on the left side than on the right in the proportion of 4:3. Schmidt, in his series, found 8 aneurysms on the right side and 13 on the left. On the other hand, Kerppola [11] found aneurysms more frequently on the right side. Fearnsides' cases were almost equally divided; 24 occurred on the left and 26 on the right side. These statistics seem to indicate that the side is not an important factor either in the production or in the diagnosis of an intracranial aneurysm.

The sex incidence of intracranial aneurysms shows the same variation. Gowers reported the disease to be more common in men than in women. In the series of 23 cases reported by Schmidt, only 6 were in men. Fearnsides, likewise, found the disease more prevalent in women.

Aneurysms may occur at any time in life. They have been known to occur during childhood, and even during infancy. Pitt [12] reported the case of a boy 6 years of age who was admitted to the hospital with acute periostitis of the femur. Death occurred one month later with symptoms of a tuberculous meningitis, and at necropsy a small ruptured aneurysm of the right vertebral artery was found. The age incidence in his series of 19 cases was very unusual. Two fifths of the patients were between the ages of 10 and 20. The high incidence in early life can probably be explained by the fact that he found a fungating endocarditis in 75 per cent of his cases. A study of the age incidence reported by various observers in series of cases which include all types of aneurysms, regardless of the etiology, leads one to the conclusion that intracranial aneurysms are most common in the second, third and fourth decade.

ETIOLOGY AND PATHOLOGY

The fundamental prerequisite for the development of an aneurysm is a localized weakness of the wall of a vessel. Turnbull [13] divided

11. Kerppola, W.: Zur Kenntnis der Aneurysmen an den Basalarterien des Gehirns, Arb. a. d. path. Inst. a .d. Univ. Helsingfors **2**:115, 1919-1921.

12. Pitt, G. N.: The Goulstonian Lectures on Some Cerebral Lesions, Brit. M. J. **1**:829, 1890.

13. Turnbull, H. M.: Alterations in Arterial Structure and Their Relation to Syphilis, Quart. J. Med. **8**:201, 1914-1915.

aneurysms into two main groups: (1) congenital and (2) acquired. The factors concerned in the formation of the latter group are inflammation, degeneration, heightened blood pressure and possibly trauma. It is often difficult to determine in which group an aneurysm belongs. The degenerative changes in the wall of the aneurysmal sac may be the result of a previous inflammatory focus or may be due to arteriosclerosis. Unless a detailed microscopic study of the wall of the aneurysm and other uninvolved vessels is made, the classification as to etiology is very uncertain.

Congenital Aneurysms.—The relatively frequent occurrence of sudden death caused by rupture of an intracranial aneurysm in otherwise young healthy persons has led to the belief that these cases are due to a congenital vascular malformation. Not infrequently intracranial aneurysms are associated with vascular anomalies in other parts of the body. Of 32 cases of coarctation of the aorta with neurologic manifestations, reviewed by Woltman and Shelden,[14] necropsy showed aneurysm with rupture in 9.4 per cent. Parker[15] reported the case of a man 21 years of age who died twelve days following rupture of an aneurysm of the left posterior communicating artery. A second aneurysm was found on the anterior communicating artery. In addition to the aneurysms, there was congenital atresia of the aorta. Parker regarded this case as an example of congenital aneurysms. Lichtenberg and Gallagher[16] recently reported a case of coarctation of the aorta associated with other vascular anomalies in which the patient presented symptoms of intermittent leakage of a cerebral aneurysm.

Forbus,[17] following a careful study of miliary aneurysms (so-called "congenital") of the cerebral arteries, concluded that these aneurysms as such are not congenital malformations. His conclusions were based on the detailed anatomic studies on both the aneurysms and the uninvolved cerebral vessels in the case of a 24 year old Negro who died three hours after the onset of a subarachnoid hemorrhage. He found complete absence of the muscular layer in the walls of 5 aneurysms, all of which occurred at the points of bifurcation of the vessels. In view of the constant location of the aneurysms in this position, microscopic studies were made of the arteries of the circle of Willis other

14. Woltman, H. W., and Shelden, W. D.: Neurologic Complications Associated with Congenital Stenosis of the Isthmus of the Aorta, Arch. Neurol. & Psychiat. **17**:303 (March) 1927.

15. Parker, H. L.: Aneurysms of Cerebral Vessels; Clinical Manifestations and Pathology, Arch. Neurol. & Psychiat. **16**:728 (Dec.) 1926.

16. Lichtenberg, H. H., and Gallagher, H. F.: Coarctation of the Aorta, Am. J. Dis. Child. **45**:1253 (June) 1933.

17. Forbus, W. D.: On the Origin of Miliary Aneurysms of the Superficial Cerebral Arteries, Bull. Johns Hopkins Hosp. **47**:239, 1930.

than those connected with the aneurysms. Sections taken from the dividing points of the two middle cerebral arteries showed a peculiar defect in the muscularis at the apex of the acute angle formed by the division of the vessel. In a series of 12 other cases of cerebral aneurysm which were studied, he found conspicuous medial defects at points of division of the cerebral vessels. Another group of cases without aneurysm was selected for control studies, and the same type of defect was frequently found. The demonstration of the defect in the muscularis in the cerebral vessels of a stillborn child seemed definitely to prove its developmental origin. Finally, Forbus was able to demonstrate experimentally that the point of maximum pressure in the wall of the vessel under normal conditions of circulation is that point corresponding to the location of the defect in the muscularis. It was his opinion that miliary aneurysms are acquired lesions resulting from a congenital muscular defect and degeneration of the internal elastic membrane due to continued overstretching.

Acquired Aneurysms.—The most frequent cause of degeneration of the arterial walls of cerebral vessels leading to aneurysmal formation is arteriosclerosis. It must be remembered that it is often a patchy disease, and unless careful histologic examination of the wall of the aneurysm is made in the absence of generalized arterial degeneration and cardiac hypertrophy, the causative factor may be overlooked. Strauss and his co-workers[18] stated: "In our experience, arteriosclerosis of cerebral vessels is the most common pathologic condition responsible for the disorganization of the vessel wall, the aneurysmal formation, the rupture and the hemorrhage into the subarachnoid space, irrespective of the age of the patient." Parker also regarded arteriosclerosis as one of the more common causes.

The second important cause of aneurysms of the acquired type is inflammation due to infection by bacteria, the mycotic aneurysms. In the series of 44 cases reported by Fearnsides, 13 cases showed cerebral aneurysms caused by infected emboli. The lesions occurred most frequently in cases of progressive malignant endocarditis (10 cases). According to this observer, the arteries are most commonly affected by impaction of emboli in their lumens.

Syphilis was formerly regarded as a common cause of intracranial aneurysms. In 1908, Bradford,[19] in his discussion of the pathology of cerebral aneurysms, said, ". . . in the case of cerebral aneurysms

18. Strauss, I.; Globus, J. H., and Ginsburg, S. W.: Spontaneous Subarachnoid Hemorrhage: Its Relation to Aneurysm of Cerebral Blood Vessels, Arch. Neurol. & Psychiat. **27**:1080 (May) 1932.

19. Bradford, J. R.: Certain Aneurysms of Cerebral Vessels, Lancet **2**:703, 1908.

their causation by syphilis is, if anything, a more definite characteristic than it is even in such aneurysms as those of the aorta." The more recent writers, however, feel that syphilis plays a minor rôle in the production of intracranial aneurysms. Fearnsides was unable to find a single case due to this cause in his series of 44 cases. Turnbull [20] likewise reported that he had not encountered an aneurysm of a cerebral vessel due to syphilitic inflammation in 10,500 autopsies. In 4 cases of intracranial aneurysm with cerebrospinal syphilis Schmidt was unable to prove that the aneurysm was caused by syphilis. It was impossible to demonstrate that the aneurysm found in case 3 of this series was definitely due to syphilis, although both the blood and the spinal fluid gave a positive reaction. There is sufficient evidence, however, to regard syphilis as a rare cause of intracranial aneurysm. Saathoff [21] and others pointed out that the basilar artery is more commonly affected by syphilitic inflammation than other vessels at the base of the brain. The reason for this has been attributed to its larger size. Krabbe and Backer [22] reported a large aneurysm of the basilar artery in a man 40 years of age with definite evidence of syphilis. The microscopic examination showed the vessels of the connective tissue wall of the aneurysm to be surrounded by plasma cells and lymphocytes. The meningeal and medullary vessels also showed cellular infiltration. Globus and his co-workers demonstrated a gummatous process in the vasa vasorum in the basilar artery in one of their cases of subarachnoid hemorrhage presumably due to the rupture of a small aneurysm. Shore [23] reported 2 cases of intracranial aneurysm in which the correct diagnosis was made during life, and was later verified at necropsy. In this report he mentioned the case of a woman under his observation who died from uremia. At necropsy, 4 aneurysms of intracranial arteries were found. The cellular reaction in the wall of the aneurysms and a 4 plus Wassermann reaction of the spinal fluid suggested a syphilitic etiology. It would seem, therefore, that syphilis may be regarded as a rare cause of intracranial aneurysms, especially those which occur on the basilar artery.

Trauma, while a frequent cause of arteriovenous aneurysms within the cranium, is rarely the cause of the type of aneurysm under discussion. It is difficult to trace the causal relationship in cases in which an aneurysm is found with a previous history of fracture of the skull or injury to the brain.

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20. Turnbull, H. M.: Intracranial Aneurysms, Brain **41**:50, 1918.

21. Saathoff, B.: Beitrag zur Pathologie der Art. basilaris, Deutsches Arch. f. klin. Med. **84**:384, 1905.

22. Krabbe, K. H., and Backer, K. H.: Contributions au diagnostic des anévrismes de l'artére basilaire du cerveau, Acta med. Scandinav. **56**:95, 1922.

23. Shore, B. R.: Intracranial Aneurysms, Arch. Neurol. & Psychiat. **21**:607 (March) 1929.

CLINICAL MANIFESTATIONS

Aneurysms of the circle of Willis may exist for many years without producing symptoms during life, and the aneurysm is discovered accidentally at necropsy. On the assumption that an adequate history is obtained, the number of cases comprised in this group is probably small. These cases are of little interest from a clinical standpoint, owing to the impossibility of making a diagnosis during life. The great majority of intracranial aneurysms produce a rather characteristic symptomatology, especially after rupture has taken place. Symonds distinguished three groups of clinical signs and symptoms: (1) those due to mechanical pressure on neighborhood structures; (2) those peculiar to the aneurysm itself—sudden leakage from the sac—and (3) those due to the disease which was the primary cause.

Neighborhood Signs.—The site of the aneurysm is of primary importance in determining the focal signs. Aneurysms arising from the intracranial portion of the carotid artery, the proximal portions of the middle cerebral artery and the circle of Willis at the point of bifurcation of these vessels constitute the anterior group. Aneurysms involving these vessels produce a clinical picture which is remarkably constant. Owing to the higher evidence of aneurysms on the carotid portion of the circle of Willis and the constancy of the clinical pictures, the aneurysms occurring in this location are diagnosed more frequently than elsewhere.

Anterior Group.—The focal signs and symptoms which may occur before rupture takes place differ in no way from those produced by other expanding lesions of the middle fossa. Pain localized in the supraorbital region on the same side may exist for months or years before the focal signs appear. With increase in the size of the aneurysm, other symptoms develop which are the result of direct pressure on contiguous structures: intermittent sharp shooting pains over the upper portion of the face, ptosis, diplopia and impairment of vision. The neighborhood symptoms may develop gradually as in tumor of the brain, but more characteristic is the sudden development with leakage from the sac resulting in the formation of a false aneurysm. Cushing [24] described the syndrome clearly:

> . . . an aneurysm should always be considered in a differential diagnosis when an apoplectic attack or series of attacks of comparatively sudden onset is followed by symptoms pointing to the region of the internal carotid in its intracranial portion, namely, a unilateral oculomotor palsy with ptosis and occipitofrontal pain with lowered sensitivity of the upper trigeminal field.

The papers by Albright, Parker, Pfingst and Spurling and Symonds describe in detail the focal signs produced by aneurysms arising from

24. Cushing, H.: Contributions to the Study of Intracranial Aneurysms, Guy's Hosp. Rep. **73**:159, 1923.

the carotid portion of the circle of Willis. The oculomotor nerve is involved more frequently than any other cranial nerve. In the series of 5 cases reported in this paper there was paralysis of this nerve in every case. Next in frequency is the involvement of the sensory portion of the trigeminal nerve, which is manifested by sharp neuralgic pains over the face, especially in the supra-orbital region. The motor division of the trigeminal nerve, olfactory, optic, trochlear and abducens nerves are less frequently involved.

It is often difficult to correlate the results of the clinical examination of the optic nerves with those of the postmortem examination. Aneurysms of the carotid portion of the circle of Willis frequently compress and stretch the optic nerve on the side of the aneurysm, yet during life there is little evidence of disturbed function. Beadles called attention to the rarity of optic neuritis and atrophy. Fearnsides, likewise, was unable to prove that optic neuritis existed in any of his cases previous to rupture. He also stated that aneurysms of syphilitic origin are more frequently associated with fundal changes, but the evaluation of the relative importance of the meningitis and aneurysmal formation was difficult. This problem was encountered in case 3 of this series in which optic atrophy and active chorioretinitis were found on the same side as the aneurysm. The visual fields are sometimes altered, and practically every type of defect has been described. Bramwell [25] described a case in which a bitemporal hemianopia occurred. Packard and his co-workers [26] reported a case in which an aneurysm of the right middle cerebral artery infringed on the right optic nerve anterior to the chiasm with resultant obliteration of the nasal field of the right eye. Kirby [27] reported irregular contraction of the right visual field, diminution of visual acuity and pallor of the optic disk in a case of aneurysm of the right internal carotid artery. In a case studied by Schmidt the defect of the visual field was a central scotoma.

Palsy of the cranial nerves may be accompanied by symptoms due to compression of other neighboring structures. In aneurysms arising from the internal carotid artery and its branches, the cerebral peduncle may be compressed, resulting in signs of involvement of the pyramidal tract on the contralateral side of the body. As the oculomotor nerve passes on the mesial aspect of the peduncle, the syndrome of Weber is common. Aneurysms involving the anterior cerebral and anterior communicating arteries are less likely to produce palsy of the cranial nerves.

25. Bramwell, B.: Intracranial Aneurysms and Meningeal or Extra-Cerebral Hemorrhage, Clin. Studies, Edinburgh **4**:289, 1906.

26. Packard, M., and Zabriskie, E. G.: Basal Cerebral Hemorrhages, J. A. M. A. **85**:1633 (Nov. 21) 1925.

27. Kirby, D. B.: Aneurysm of the Intracranial Portion of the Internal Carotid Artery, Am. J. Ophth. **7**:577, 1924.

Large aneurysms located anteriorly may compress both peduncles, the midbrain and even the pons.

Posterior Group.—The syndrome produced by aneurysms of the basilar and vertebral arteries and their branches is not as uniform as that produced by the anterior group. The seventh, eighth, ninth, tenth and twelfth cranial nerves may be involved owing to direct compression. The third nerve may be compressed by an aneurysm situated anteriorly at the bifurcation of the basilar artery. Perhaps the most common finding is weakness or paralysis of the extremities, caused by compression of the pyramidal tracts in the brain stem. This may take the form of recurrent attacks of hemiplegia on one side, or the hemiplegia may occur alternately, first on one side and then the other side. The pyramidal tracts may be compressed simultaneously, producing a paraplegia. Bulbar symptoms manifested chiefly by dysarthria and dysphagia are not uncommon. Thrombosis of the basilar artery due to syphilitic endarteritis may produce a similar picture.

SIGNS AND SYMPTOMS DUE TO RUPTURE OR LEAKAGE

The characteristic symptomatology of aneurysms begins when rupture occurs. Premonitory symptoms of an indefinite character may precede rupture for several days. The rupture of the aneurysm is usually manifested by a sudden apoplectic seizure with loss of consciousness, vomiting and sometimes a convulsion. Death may occur at this time. If fatal hemorrhage does not occur, the patient gradually regains consciousness and complains of severe fronto-occipital headache, stiffness of the neck and perhaps pains in the extremities, due to irritation of the meninges by the extravasation of blood into the subarachnoid space. The clinical picture resembles a fulminating septic meningitis. Frequently the focal signs, notably palsy of the third nerve, appear at this time. The preexisting focal signs are increased owing to the formation of a clot around the aneurysm.

The neurologic picture at this stage is of interest. Signs of meningeal irritation can be elicited, stiffness of the neck and the Brudzinski and Kernig signs. The meningeal signs, as a rule, are not as pronounced as one finds in inflammation of the meninges due to bacterial invasion. In some cases with massive extravasation of blood into the subarachnoid space the signs of meningeal irritation may be minimal or entirely absent. Fever and light leukocytosis are almost constant findings. Hemiplegia, either transient or permanent, which occurs so frequently at the time of rupture of the aneurysm, may be due to several different causes. The aneurysm may rupture into the parenchyma of the brain and even into the ventricular system, producing considerable destruction of tissue. Again, intracerebral hemorrhages may occur in parts of the brain far

removed from the site of the ruptured aneurysm. Hassin [28] reported the clinicopathologic observations in a case of this type in which a ruptured aneurysm of the posterior communicating artery was found associated with multiple hemorrhages in the region of the ipsilateral basal ganglions. He attributed the intracerebral hemorrhages to rupture of veins caused by a local fall in the arterial blood pressure at the time of rupture of the aneurysm at the base. Sands [29] described a similar case in which hemorrhages were found in the pons and the midbrain in a case of ruptured aneurysm of the posterior communicating artery. The delayed and also the gradual onset of the hemiplegia in some cases following rupture of the aneurysm may be due to thrombotic occlusion of the involved vessel rather than to hemorrhage.

The ophthalmoscopic picture before rupture, as mentioned previously, is in no way characteristic. However, following rupture, certain abnormalities may be observed which are the result of extravasation of blood along the sheath of the optic nerve. The most characteristic and important, from a diagnostic standpoint, is the presence of a diffuse hemorrhage adjacent to the optic disk. Other smaller hemorrhages may be seen in the more peripheral portions of the retina which are not unlike those seen in other conditions. The veins are usually engorged and slight edema of the disk may be present, but it is rare to have a high degree of choked disk. Riddoch and Goulden [30] believe that the intraocular hemorrhage which occurs in these cases is due to venous obstruction.

Examination of the spinal fluid immediately following the rupture of an aneurysm reveals a uniformly bloody spinal fluid under increased pressure. Microscopic examination shows red and white blood cells in the same proportion in circulating blood. On centrifugation, the supranatant fluid is colorless, and the sediment is composed of red blood cells. If the lumbar puncture is delayed for a day or two following the apoplectic seizure, the fluid is xanthochromic and the red cells are found in various stages of disintegration. The spinal fluid may be quite normal two weeks after a single subarachnoid hemorrhage.

Associated Disease.—In addition to the signs and symptoms resulting from the aneurysm and its rupture, there may be added the symptoms and signs of the underlying disease. The most uniform clinical picture of the associated disease is that produced by bacterial endocarditis: anemia, fever, leukocytosis, pains in the joints, cardiac murmurs, hematuria

28. Hassin, G. B.: The Pathogenesis of Cerebral Hemorrhage, Arch. Neurol. & Psychiat. **17**:770 (June) 1927.

29. Sands, I. J.: Aneurysms of Cerebral Vessels, Arch. Neurol. & Psychiat. **21**:37 (Jan.) 1929.

30. Riddoch, G., and Goulden, C.: On the Relationship Between Subarachnoid and Intra-Ocular Hemorrhage, Brit. J. Ophth. **9**:209, 1925.

and other embolic phenomena. The sudden appearance of meningeal signs in the course of a subacute bacterial endocarditis should always make one suspicious of bleeding from a mycotic aneurysm. Focal signs of aneurysm in these cases are frequently absent, owing to the fact that mycotic aneurysms have a tendency to rupture before attaining large size. Meningeal signs may also occur in this disease owing to bacterial invasion of the meninges, resulting in a purulent meningitis. This, however, is an extremely rare complication. Winkelman and Eckel,[31] from a study of the cerebral complications of subacute bacterial endocarditis, commented on its infrequent occurrence.

The signs and symptoms of generalized arteriosclerosis, hypertension, cardiovascular anomalies and perhaps syphilis may complicate the picture.

COURSE

Cerebral aneurysm may exist for many years without causing symptoms and may be discovered at postmortem examination, death being the result of some intercurrent disease. Again, sudden death may occur at the time of the first leakage. The most characteristic clinical course occurs in those cases with recurrent leakage accompanied by symptoms and signs of meningeal irritation. The intervals of freedom from symptoms following the first attack may be days, months or years.

DIAGNOSIS

The diagnosis of intracranial aneurysm cannot be made with any degree of certainty until rupture occurs. Focal signs, if present before rupture takes place, are not different from those found in other localized disease processes within the cranium, notably cerebral tumor. Furthermore, Fearnsides, Cushing and others have pointed out that cerebral aneurysms which do not rupture rarely give rise to symptoms. The uniform clinical picture produced by aneurysms arising from the anterior portion of the circle of Willis makes the diagnosis during life possible. The syndrome is described clearly by Cushing in his discussion of Symonds' report:

> An aneurysm should always be considered in a differential diagnosis when an apoplectic attack or series of attacks of comparatively sudden onset is followed by symptoms pointing to the region of the internal carotid in its intracranial portion, namely, a unilateral oculomotor palsy with ptosis, and occipito-frontal pain with lowered sensitivity of the upper trigeminal skin field. Should there be in addition subhyaloid retinal hemorrhages, and should the cerebrospinal spaces be found to contain free blood (or possibly even xanthochromia in the intervals between periods of leakage) a diagnosis, as the author makes clear, is reasonably certain.

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31. Winkelman, N. W., and Eckel, J. L.: The Brain in Bacterial Endocarditis, Arch. Neurol. & Psychiat. **23**:1161 (June) 1930.

If focal signs exist before rupture occurs, it may be difficult to make a differential diagnosis between aneurysm and tumor arising from structures located in the middle fossa. However, in the presence of hypertension and the absence of signs of increased intracranial tension, the possibility of aneurysm should be strongly considered. Roentgenograms of the skull may be of some help. Sosman described changes in the roentgenograms which permit a diagnosis in about 25 per cent of the cases of aneurysm involving the internal carotid artery. When rupture with subarachnoid bleeding occurs, the diagnosis of tumor can usually be excluded. Bleeding into the subarachnoid space, due to hemorrhage from a tumor, is regarded as being extremely uncommon. Strauss and his co-workers encountered subarachnoid hemorrhage in only 1 case in a series of 150 verified tumors of the brain during the preoperative course. It has likewise been the experience in this clinic that this complication of cerebral tumor is exceedingly rare. The presence or absence of a cranial bruit is not an important differential finding. It has been frequently stated that head murmurs occur more often in diseases other than aneurysm.

Sudden onset of symptoms and signs of meningeal irritation with headache, stiffness of the neck, fever and leukocytosis at once suggests the possibility of purulent meningitis. A lumbar puncture in cases of subarachnoid hemorrhage will reveal the presence of blood. The ratio of white to red blood cells in the spinal fluid may be slightly higher than one finds in circulating blood, owing to the meningeal reaction to the presence of a foreign substance in the subarachnoid space. The number of white blood cells in the spinal fluid is small compared to the number found in cases of hemorrhagic meningitis, owing to bacterial inflammation.

The clinical picture with headache, fever, ocular palsies, mental clouding and meningeal signs may simulate acute epidemic encephalitis. Examination of the spinal fluid, however, is of great value in making the differential diagnosis. While the presence of blood is not unheard of in acute epidemic encephalitis, it is unusual. McIntyre [32] recently reported a series of 20 cases of an unusual form of encephalopathy, probably of infectious origin, in which hemorrhage into the spinal fluid occurred in 13 of the 20 cases. In these encephalitides, however, there is an increase in the number of lymphocytes and in the amount of globulin which is not found in cases of subarachnoid hemorrhage without inflammation.

Intracerebral hemorrhages which rupture into the ventricle may closely simulate the picture produced by rupture of a basilar aneurysm.

32. McIntyre, H. D.: An Unusual Encephalopathy, Probably Infectious in Origin: Clinical Report on Twenty Cases, J. A. M. A. **100**:1097 (April 8) 1933.

The presence of palsy of the cranial nerves would favor the latter diagnosis.

The diagnosis may also be confused with cerebrospinal syphilis, hemorrhagic pachymeningitis and ophthalmoplegic migraine. Serologic studies and examinations of the spinal fluid are helpful in making a diagnosis of a basilar syphilitic meningitis the clinical picture of which may be similar to that of basilar aneurysms. It is quite possible that in some cases diagnosed as recurrent ophthalmoplegic migraine the symptoms are due to small basilar aneurysms with recurrent leakage.

TREATMENT

The treatment in these cases in the past for the most part has been merely palliative. Repeated lumbar puncture with the careful withdrawal of fluid, in order to relieve increased intracranial pressure, has been advised by several observers. This procedure, however, is not without danger. The disturbance of pressure relations within the cranium may result in increased bleeding from the aneurysm, and in some instances fatal hemorrhage has occurred. Surgical treatment, consisting of ligation of the internal carotid artery, has been used in a few cases in which an accurate diagnosis was possible. The value of this procedure is doubtful. Ligation of the internal carotid artery may produce a hemiplegia, but if one remembers that this is a frequent complication of untreated aneurysm it is worthy of further trial in an attempt to avoid fatal rupture.

News and Notes

UNIVERSITY NEWS

Dr. Judah L. Magnes, Chancellor of the Hebrew University in Jerusalem has announced the proposed nomination of Prof. Ludwig Halberstaedter as head of a new department of radiobiology at the University. Professor Halberstaedter, who is now in charge of the Station for Cancer Therapy at the Rothschild Hadassah Hospital in Jerusalem, is internationally recognized as an expert in the treatment of malignant tumors. Other appointments to be proposed for the new Department of Cancer Research include that of Dr. H. A. Krebs, formerly of the Kaiser Wilhelm Physics Institute, Berlin-Dalhem, who is now at the University of Cambridge, England, and that of Dr. Leonid Doljansky, formerly of the University Institute of Pathology of Berlin, who is now at the University of Copenhagen. For the first time since the founding of the Hebrew University, the government of Palestine is supporting three research projects, including the control of malaria and fowl pox.

SOCIETY NEWS

International Association for Prevention of Blindness.—The International Association for the Prevention of Blindness held its annual meeting in Paris, France, on May 14. Agreement on the classification of the various causes of blindness was sought by the American and European delegates. Prof. F. de Lapersonne of the University of Paris, president of the association, pointed out the importance of statistics on the causes of blindness intelligible to the layman. Dr. Park Lewis of Buffalo, vice president of the association, urged that an effort be made to classify the extent or degree of blindness when vision exists but not in sufficient amount to be of practical use. In a report submitted by Lewis H. Carris of New York, managing director of the American National Society for the Prevention of Blindness, fifty different etiologic classifications of blindness were listed. Among the major causes of blindness included in the American report are the following: infectious diseases, such as diphtheria, measles, ophthalmia neonatorum, scarlet fever, syphilis, trachoma and tuberculosis; traumatic and chemical injuries sustained in war; industrial accidents; traffic accidents and surgical procedures, and congenital and hereditary causes.

Colorado Ophthalmological Society.—The twelfth annual summer graduate course in ophthalmology and otolaryngology will be given under the auspices of the Colorado Ophthalmological Society in Denver from July 23 to Aug. 4, 1934. For the convenience of those wishing to enroll for only one branch of the work, the first week will be devoted to ophthalmology and the second to otolaryngology. The fee for the course is $50. Those taking ophthalmology or otolaryngology only will be required to pay a fee of $30. Fees must be sent at the time of registration, and checks should be made pay-

able to H. L. Whitaker, Treasurer, and mailed to him at 1234 Republic Building, Denver. Because of a limited registration of sixty-five, applicants for the course are requested to register as early as possible.

German Ophthalmological Society.—The meeting of the society will take place in Heidelberg on Aug. 6 to 8, 1934, in the Town Hall. On the evening of August 5, a reception will be held on the terrace of the Europäischer Hof.

Association for Research in Ophthalmology.—The fifth scientific meeting of the association will be held on June 12, 1934, in Cleveland in the Hotel Allerton. Members of the Section of Ophthalmology of the American Medical Association are invited to attend, and should send their names and addresses to Dr. Conrad Berens, Secretary-Treasurer, 35 East Seventieth Street, New York.

All India Ophthalmological Society.—The annual conference was held at the new All India Institute of Hygiene, Calcutta, on Dec. 19 to 21, 1933. An exhibit of slides and motion pictures illustrating the care of the eyes and the need for preventive work were furnished by the Association for the Prevention of Blindness in Bengal. Colonel Duggan was elected president. The 1935 meeting will be held in Madras.

GENERAL NEWS

St. John Ophthalmic Hospital, Palestine.—During 1933 the Order of St. John supervised ten ophthalmic clinics in Palestine, and it has planned for the training of young Arab girls in hygiene and home nursing. During the year, the staff of the St. John Ophthalmic Hospital in Jerusalem was increased to four ophthalmologists; the total attendance numbered 89,895, the in-patients 1,179 and the operations 3,630.

Abstracts from Current Literature

EDITED BY DR. WILLIAM ZENTMAYER

Anatomy and Embryology

EXPERIMENTAL PRODUCTION OF CYCLOPEAN CHICKS. E. WOLFF, Compt. rend. Soc. de biol. **114:** 1030, 1933.

Cyclopia occurs spontaneously in birds with extreme rarity. Until now, embryologists have not succeeded in producing these monsters among amniotes (reptiles, birds and mammals). However, by utilizing the local irradiation method of Ancel and Wolff, various types of cyclopia were obtained with considerable regularity, according to the period of development, the localization, and the extent of the intervention. Sixty-three chicks with cyclopia were demonstrated, forty of which had lived more than ten days. Irradiation of the anterior end of the neural groove within from eighteen to twenty-two hours after incubation produced rudimentary one-eyed median cyclopia. Irradiation of embryos beyond the two somite stage resulted in single orbit cyclopia, with the two eyes more or less developed.

J. E. LEBENSOHN.

Conjunctiva

SIMILARITY OF DISEASES OF THE EYE IN VERNAL CATARRH AND PHOTO-OPHTHALMIA CAUSED BY ULTRAVIOLET RAYS. I. A. KORENEVITCH, Sovet. vestnik oftal. **2:** 389, 1933.

Korenevitch studied the clinical pictures of four cases of vernal catarrh and six cases of corneal diseases caused by ultraviolet rays. In both groups of cases the slit lamp showed numerous grayish-white flat scales located in the superficial layers of the cornea in its upper half and on the nasal side of the palpebral fissure. The scales were stained with fluorescein. He believes these changes to be erosions of the cornea and the scales to be degenerated or necrotic epithelium.

He comes to these conclusions: 1. Vernal catarrh is a disease not only of the conjunctiva but also of the cornea. This is demonstrated by the presence of photophobia and epiphora and by the results of studies with the slit lamp. 2. The disease of the cornea in vernal catarrh is closely related to photo-ophthalmia due to the action of ultraviolet rays. 3. Since the clinical pictures are similar, these diseases must have the same etiology. 4. Ultraviolet rays cause vernal catarrh in persons with certain constitutional and physiologic idiosyncrasies. The study of persons suffering from vernal catarrh may aid in the professional selection of those predisposed to photo-ophthalmia. 5. As vernal catarrh is a chronic disease, it presents better material for study than does the transitory photo-ophthalmia.

O. SITCHEVSKA.

Comparative Ophthalmology

THE DORSAL EYES OF CERTAIN LAND MOLLUSKS. A. LABBÉ, Compt. rend. Soc. de biol. **114:** 1002, 1933.

Besides the usual eyes characteristic of invertebrates, many Onchidiidae possess the dorsal eyes characteristic of vertebrates. This development is due to a differentiation of special "optoblastic" tissue which arises by division of epidermal cells and forms in the depths of a cutaneous invagination a spherical mass, from which a simple or stratified retina develops. The crystalline lens results from the glassy secretion of a silicious gland.

J. E. LEBENSOHN.

MORPHOLOGIC, CHEMICAL AND HISTOCHEMICAL STUDY OF THE GLAND OF HARDER. A. STILO, Ann. di ottal. **61:** 453 (June) 1933.

The gland of Harder, which is present in all animals with a third eyelid, was studied in the rabbit. It is divided into a red-gray portion and a smaller white portion, each with separate ducts. The former contains much lipoid material and secretes a fatty material which facilitates movements of the third lid over the globe. The latter contains less lipoid and secretes a fluid consisting chiefly of mucin and water, which seems to function like tears in these animals, which possess only rudimentary lacrimal glands. The intimate arrangement of capillaries in a plexus around each glandular acinus suggests that an internal secretion may also be formed having some function with respect to the visual apparatus. A rudimentary Harder gland is present in the human black race.

S. R. GIFFORD.

Cornea and Sclera

A SERIES OF ONE HUNDRED CASES OF HYPOPION ULCER, WITH SPECIAL REGARD TO A METHOD OF TREATMENT. A. C. REID, Brit. J. Ophth. **17:** 162 (March) 1933.

In this series of cases a modified Wessely cautery was used. The cautery is heated to an optimum of from 75 to 85 C. (similar to the Shahan thermophore). The point of the cautery is stroked over the surface of the ulcer, the edges where advance seems likely being carefully explored. The cautery even breaks through into any fresh focus without attacking the secondary deep infiltration abscess, if present.

The author believes that every patient seen within twenty-four hours of injury with an ulcer that has an appearance of being infected should, when possible, be admitted to the ward.

The author has abandoned the use of the red hot cautery. By considering the severe and even hopeless cases along with the less severe and even mild cases, the series shows "good" results in sixty-five.

W. ZENTMAYER.

COMPARATIVE HISTOLOGIC STUDY OF THE PRINCIPAL METHODS OF CORNEAL TATTOOING. O. BARATTA, Arch. di ottal. **40:** 180 (April) 1933.

Baratta reviews the results of tattooing with india ink and the various more recent chemical methods of staining (without mentioning, how-

ever, work by Gifford and Steinberg and by Pischel). He produced corneal scars in rabbits by scarification and after from thirty to forty days stained these scars by various methods. He removed the eyes for section after one, three, six, twenty-five and thirty days. He reports on the gross staining, the inflammatory reactions and the relative stability of the stain.

One method employed was that recently described by Jasiński, according to which, after removal of the epithelium, from 1 to 5 per cent subacetate of lead was applied for thirty seconds. After irrigation this was reduced by application of from 5 to 10 per cent sodium hyposulphite (*polisulfuro di sodio*) for thirty seconds. This resulted in rather severe chemical burns of the cornea, which extended beyond the denuded area. A chestnut brown was obtained, but this faded almost completely within a few days. Sections showed an intense inflammatory reaction of the cornea, with scattered granules of pigment between the corneal lamellae. When 1 per cent silver nitrate was employed, reduced by a minimum amount of 5 per cent sodium hyposulphite, a stain varying from black to dark brown was produced, which remained almost unchanged for thirty days. A rather marked inflammatory reaction was produced, which subsided within a few days. After five days sections showed a sharply demarcated zone of brown under the corneal epithelium and smaller collections of colored granules in the anterior half of the substantia propria between the lamellae.

Eyes treated by injecting india ink into the cornea showed a severe reaction, with scattered and unsatisfactory staining.

With 2 per cent platinum chloride, applied for two minutes and reduced by 2 per cent hydrazine hydrate, a lustrous black was produced, with only slight inflammatory changes. A zone of diffuse corneal staining beneath the epithelium was seen in sections, the fixed cells having assumed the color. There were black granules in the deeper layers of the cornea. With 2 per cent gold chloride the same diffuse staining was produced, with no other changes in the corneal cells. With a combined method, using 2 per cent gold chloride for thirty seconds and 1 per cent silver nitrate for ninety seconds, the results were about the same, but the stain was less diffuse after thirty days.

Baratta concludes that good cosmetic results may be obtained without danger to the eye by the chemical methods, especially those employing gold, platinum and the combination of gold and silver. The results are superior to those with india ink and there are fewer harmful changes in the eye.

S. R. GIFFORD.

CHEMICAL METHOD OF CORNEAL TATTOOING FOR PRODUCTION OF A BROWN COLOR. F. SPINELLI, Arch. di ottal. **40: 266** (June) 1933.

The author reviews the literature on chemical staining of the cornea, most of which is devoted to imitating the black pupil. Since in staining with gold chloride the intensity of color depends on the activity of the reducing agent employed, the author searched for a reducing agent weaker than hydrazine hydrate while of the same general properties. This he found in hydrazine hydrochloride. By varying the strength of the gold chloride and hydrazine hydrochloride solutions in the test tube, a range of colors could be obtained from dark brown with 4 per cent

gold chloride and 4 per cent hydrazine hydrochloride to light brown, resembling the shade of a hazel iris, with 4 per cent gold chloride and 2 per cent hydrazine hydrochloride. Still lighter tones were produced by further dilution of the reducing agent.

In a group of rabbits the right eyes were stained by the second method and the left eyes by the first method, the gold solution being applied for two minutes after removal of the central corneal epithelium and the reducing agent for one minute. A uniform light brown stain was produced with the more dilute reducing agent, and a darker brown with the more concentrated solution. The epithelium formed over the stained area in from three to six days, and the reactions were minimal.

Sections of eyes removed after one, three, seven, twenty and thirty-five days showed that the fine granular precipitate of brown particles occurs first in Bowman's membrane and then migrates farther back into the corneal stroma. The granules in the more posterior layers become clumped in larger particles. There was only a slight infiltration of the deeper portions of the cornea with eosinophil cells which contained brown granules.

The method was employed in two cases of corneal scars in patients with brown irides. In the first case the central portion of the cornea had been stained with platinum chloride and hydrazine hydrate. The surrounding portion of the cornea was stained with 4 per cent gold chloride and 2 per cent hydrazine hydrochloride five months later, and five months after this operation a shiny brown color was present which contrasted well with the black central stain.

In the second case a scar in the lower quadrant was stained with 4 per cent gold chloride and 2 per cent hydrazine hydrochloride. Ten months later the brown color remained unchanged and was almost identical with that of the healthy eye. S. R. GIFFORD.

PARTIAL CORNEAL TRANSPLANTATION. V. P. FILATOFF, Sovet. vestnik oftal. **2**: 136, 1933.

Professor Filatoff has done successful keratoplastic work in the Union of Soviet Socialist Republics. For prevention of prolapse of the vitreous the author is using a narrow ivory plate which is put into the anterior chamber after the incision with Graefe's knife. This plate has a hole on each end for sutures by which it is held in place. The author also uses the plate in those cases in which the lens is present. He extracts the cataractous lens after the transplant is becoming permanently transparent. If the lens is injured during the operation, the plate is removed, the lens is extracted and the plate is put immediately back into the anterior chamber, as it prevents prolapse of the vitreous. If the vitreous is liquefied it may push out both the plate and the transplant. In these cases Filatoff uses vertical and horizontal sutures at the edges of the leukoma and ties them evenly after the transplant is put in place. Thus the sutures form a net over the transplant which prevents its movement forward or backward.

In five cases of staphyloma with predisposition to glaucoma, in which a successful transplantation was doubtful, the author did a preliminary operation of removal of the calcified scar tissue with the adhesions. The technic is described. In one case an infection devel-

oped, in the second the eyeball became atrophic, and in three cases the result was good. The author also suggests that the cut away scar tissue be substituted with healthy corneal lamellae in order to create a favorable field for the future transplant. He describes the technic of this operation.

Filatoff recommends the use of warm applications to the transplant of chicken embryo juice in the postoperative period for prevention of opacification of the transplant. The idea of this method is to improve the resistance of the corneal epithelium by the unspecific activators of the embryonal juice. It is analogous to the theory of culture of tissue. The author is experimenting in this field at present.

O. SITCHEVSKA.

Experimental Pathology

OXYPHILOUS FORMATIONS RESEMBLING NEGRI BODIES IN THE CORNEAL EPITHELIUM OF RABIC RABBITS. C. LEVADITI and R. SCHOEN, Compt. rend. Soc. de biol. **114:** 501, 1933.

Certain filtrable viruses, such as those associated with poliomyelitis, encephalitis, herpes and rabies, tend to affect especially ectoderm (cornea, skin or mucous membrane) or neuro-ectoderm (peripheral and central nervous system). The Negri bodies represent the visible phase of the rabies virus. So far they have not been found in skin or mucous membrane, but in seven of eleven rabbits subjected to intraocular inoculation similar bodies were demonstrated in the corneal epithelium. This experimental finding thus tends to support the conception of "neurotropic ectodermoses."

J. E. LEBENSOHN.

EXPERIMENTAL STUDY OF RETINAL CHANGES IN THE HUMAN EYE EXPOSED TO INTENSE LIGHT. L. MAGGIORE, Ann. di ottal. e clin. ocul. **61:** 81 (Feb.) 1933.

Three eyes destined to enucleation because of orbital tumor were exposed to bright focal light and were then studied histologically. In one a beam of solar light was focused in the dilated pupil for ten minutes, while in the other two the light of a Zeiss-Hammer lamp was used for thirty minutes. Edema of the corneal epithelium prevented ophthalmoscopic examination of the retina before enucleation, which was performed almost immediately. Fixing fluid was injected into the eye immediately after enucleation.

Exposure to solar light for ten minutes produced a slight edema and hyperemia of the choroid and an edema of the retina affecting especially the outer plexiform and nerve fiber layers. The layer of rods and cones was adherent to the pigmentary epithelium, so that parts of the latter were pulled away from the underlying choroid. This was considered an exaggeration of the protective action of the pigmentary epithelium in bright light.

Exposure to the light of a Zeiss-Hammer lamp for thirty minutes gave rise to more marked changes. The rods and cones showed marked degeneration, especially in the central portions of the retina. The macular area was much thickened, owing to dissociation of the outer plexiform

layer into a network of fibrils. The rods and cones lost their staining properties, and their outer elements were reduced to a mass of refractile granules. The more marked changes in the macular area are to be explained as due only partially to the refracting power of the dioptric media, since the source of light was so close to the eye that a large area about the macula was equally irradiated. The author believes that the anatomic structure of the macula, with its highly developed external plexiform layer and peculiar blood supply, must be considered of great importance in the changes observed, which correspond to the ophthalmoscopic picture observed by others in eyes exposed to excessive light.

Similar changes were produced in dogs, while in rabbits, in which the anatomic conditions are much more unlike those in man, only slight retinal changes occurred.

Since the experiments of Cirincione and Ciaceri with thermo-electric terminals showed that the temperature of the intra-ocular structures was not changed appreciably by external irradiation, Maggiore believes that the changes which he observed must be due to the luminous rays alone.

S. R. GIFFORD.

COMPARISON OF THE EFFECT OF VARIOUS OPERATIONS FOR DETACHMENT OF THE RETINA DEMONSTRATED BY EXPERIMENTS ON ANIMALS. A. VON SZILY and H. MACHEMER, Klin. Monatsbl. f. Augenh. **90:** 806 (June) 1933.

The authors give an exhaustive description of their experiments on rabbits' eyes with various operative methods for detachment of the retina, and of the histologic examination of the globes. Thirty-one illustrations of microscopic specimens and pictures of the fundus are added. The operative and histologic technic is given, and chapters are devoted to retinal operations by puncture with the thermocautery, cauterization with the caustic rod, diathermy and electrolysis. In discussing their results, the authors render detailed accounts of the changes and effects produced by the operative procedures on the sclera, vitreous, choroid and retina, including the attachment of the retina to its substratum.

Due consideration is given the complications and dangers of the divers operations. They consist in intra-ocular hemorrhages, degeneration of the retina, with subsequent perforation and detachment after the operative procedure, and formation of cords in the vitreous. Summing up, the authors found that thermopuncture produces firm adhesions of limited extension; multiple cauterizations with the caustic, applied for a short space of time, result in subtle but spacious adhesions; large agglutinations, furthermore, may be obtained by multiple electropuncture with an adequately strong current; here single punctures with diathermy lead to limited adhesions, with electrolysis to spacious attachments. Untoward complications are unavoidably possible in the use of every method, no matter how skilled the operator or how correct the dosage may be. Only experienced operators, therefore, should undertake operative procedures for detachment of the retina. Experience can be gained only by experiments on animals.

K. L. STOLL.

General Diseases

OCULAR DISTURBANCES IN THE COURSE OF TWO LEUKEMIC SYNDROMES. BEAUVIEUX and BESSIÈRE, Arch. d'opht. **50: 377** (June) 1933.

The authors present two observations of unusual orbital and ocular manifestations in the course of leukemia which demonstrate the importance of examining the blood to clarify the diagnosis and indicate the proper etiologic treatment. The first case was puzzling because the initial lesion was a puffing of the lids with signs indicative of an infection. The recent reports of R. E. Wright and C. Pascheff are cited in connection with this case. The second case presented the chief complaint of almost total loss of vision in both eyes—the loss in the right being due to vitreous hemorrhage, that in the left to a proliferative retinitis. The case is reported because of the unusual predominance of hemorrhagic retinal disturbance as the first manifestation of leukemia.

S. B. MARLOW.

MIKULICZ' DISEASE. E. PUSCARIU and D. LAZARESCU, Ann. di ottal. e clin. ocul. **61: 161** (March) 1933.

The authors report three cases of Mikulicz' disease seen during sixteen years at the clinic in Jassy, Rumania. All the patients were men, aged 30, 17 and 70 years. In one both lacrimal glands and the right parotid were enlarged. Roentgen treatments were given. In the second patient the lacrimal glands and the right sublingual gland were enlarged. Part of the larger lacrimal gland was removed and showed very hyperemic and hypertrophic glandular tissue, with some lymphocytic infiltration. The third patient showed lacrimal swellings and marked enlargement of the superior cervical glands. Sections showed that most of the lacrimal gland was replaced by small round cells, which composed the entire cervical tumor. All the patients showed a marked increase of mononuclears in the blood and eosinophilia of from 7 to 9 per cent.

S. R. GIFFORD.

General Pathology

THE ANATOMIC LESION OF NEUROPARALYTIC KERATITIS. J. DUGUET, Arch. d'opht. **50: 398** (June) 1933.

The author has had the opportunity to follow a case of neuroparalytic keratitis through to autopsy and he records in detail the clinical and pathologic observations. The patient had hemiplegia of the left side, paralysis of the fifth, sixth and seventh nerves and neuroparalytic keratitis of the left side. Autopsy disclosed a subacute syphilitic basal meningitis with secondary invasion of the fifth nerves, and a destructive hemorrhage in the region of the lenticular nucleus of the right hemisphere. Careful histologic examination revealed an interstitial inflammation of both gasserian ganglions, greater in the right than in the left. The histologic combined with the clinical and laboratory data led to the conclusion that it was syphilitic. The histologic changes in the affected eye are also described. The author takes up systematically the lesions which occur in the eye, beginning with the earliest changes which have been observed with the slit lamp by Tagawa. He believes that the occur-

rence of the bilateral involvement of the gasserian ganglion in the case observed indicates that this ganglion plays little part in the genesis of neuroparalytic keratitis. This contention is further supported by the presence of a variety of lesions in this ganglion without the occurrence of keratitis. The observations of Dandy, Robineau and Sicard, on the other hand, seem to indicate that this condition appears in some patients operated on for facial neuralgia, even though the sensation of the cornea is intact. These observations seem to indicate that there is some point in the trigeminal tract irritation of which results in keratitis. The suggestion has been made theoretically that the sympathetic fibers in the fifth nerve are responsible. To explain the great irregularity in the incidence of keratitis the author invokes the question of anatomic variation.

S. B. MARLOW.

Glaucoma

A CLINICAL NOTE ON GLAUCOMA. E. E. MADDOX, Brit. J. Ophth. **17:** 161 (March) 1933.

For about five years the author has used high frequency current in the treatment of glaucoma. It has the effect of almost immediately lowering the tension in nearly all moderately hypertensive eyes. In most cases, repeated applications are of value. The application may be made against the temporal margin of the orbit. The current should not be turned on until contact has been made with the skin. Caution should be used with inflamed eyes.

W. ZENTMAYER.

CERVICAL DIATHERMY IN GLAUCOMA. D. MARGOLIN and H. LIPOVITZ, Sovet. vestnik oftal. **2:** 159, 1933.

Believing that the pathogenesis of glaucoma lies in the vegetative system, the authors attempted to regulate the circulation of the lymph and blood by applying diathermy in primary glaucoma. They treated three patients with absolute glaucoma and ten with chronic simple glaucoma for whom operative intervention was contraindicated because of narrow visual fields. Tcherbak's method was used. One electrode was placed at the cervical and the other at the central part of the spinal column, affecting thus the cervical sympathetic ganglions and indirectly influencing the weakened tonus of the uveal vessels. The strength of the current varied from 0.5 to 0.75 Holzknecht unit. The treatments were applied every other day and numbered from fifteen to twenty.

There were considerable improvement of vision and enlargement of the visual fields in five cases. The therapeutic effect was more marked in less advanced cases. In a few cases the treatment was unstable and had to be repeated in three or four months. No case of absolute glaucoma responded to cervical diathermy.

O. SITCHEVSKA.

Injuries

REMOVAL OF NONMAGNETIC FOREIGN BODIES FROM THE VITREOUS CHAMBER: REPORT OF TWO CASES. R. FRIEDE, Klin. Monatsbl. f. Augenh. **90:** 368 (March) 1933.

In two cases, foreign bodies which were lodged in the posterior, lower portion of the vitreous chamber were removed by the author

through an incision on the equator of the sclera, parallel to the lower corneal margin. Haab's meridional incision, although presenting many advantages, would not have yielded a similar result. The equatorial incision, made beyond the ora serrata, allows access to the anterior, as well as the posterior, portions of the vitreous chamber and hence facilitates the removal of nonmagnetic chips. In case of necessity a scleral flap may be made in the upper half of the globe. Loss of vitreous must be expected in equatorial incisions; it was less than anticipated, especially in one of these cases, in which the foreign body had lodged below the posterior pole of the eye. K. L. STOLL.

Instruments

NEW LACRYMAL SAC RETRACTOR. V. P. PATEL, Brit. J. Ophth. **17:** 39 (Jan.) 1933.

This is a modification of the Muller retractor. The blades are longer and possess three blunt teeth. The shaft is curved to fit the contour of the face. A wider retraction of the skin edges is possible. The degree of this can be regulated by a screw. The difficulty of introducing the teeth, which are larger than in other models, is obviated by undermining the skin incision. The article is illustrated. W. ZENTMAYER.

Lacrimal Apparatus

DACRYOCYSTORHINOSTOMY OR DACRYOCYSTECTOMY IN OZENA? G. RUSSO-FRATTASI, Ann. di ottal. e clin. ocul. **61:** 124 (Feb.) 1933.

Owing to the atrophic condition of the mucosa in ozena, the placing of a suture for the Dupuy-Dutemps or modified Toti operation is difficult or impossible. An intranasal operation must be performed in an infected and unclean field in this condition. Russo-Frattasi concludes that simple removal of the sac is best suited to the conditions found in ozena. S. R. GIFFORD.

Lens

A CASE OF CONGENITAL CATARACT SHOWING UNUSUAL FEATURES. D. J. WOOD, Brit. J. Ophth. **17:** 158 (March) 1933.

The patient was a Swahili boy. In each eye in the center of the dilated pupil was a dense circle of capsular opacity, with a yellowish raised epicapsular spot and several smaller specks, remains of lens matter. The central dense part was marked by concentric lines, which were rather bluish and tendinous looking in the right eye. Around this central opacity was a flat, slate-colored band, notched at its inner margin and furrowed on its surface. This represented a thinner part of the capsule with an impress of the uvea through which the dark interior was dimly seen.

The central part was separated, leaving a round disk of capsule on the anterior chamber. This was later removed, resulting in a clear space through which with glasses good vision was obtained.

Microscopic examination of the disk of capsule showed the anterior surface marked by digitate wrinkles outlined by fine atoms of pigment and the posterior surface marked with concentric wrinkles with oval nuclei lying in lines corresponding to them. No nuclei belonging to the anterior capsule were visible. W. ZENTMAYER.

THE RÔLE OF CALCIUM IN NAPHTHALENE CATARACT. M. C. BOURNE and D. A. CAMPBELL, Brit. J. Ophth. **17**: 220 (April) 1933.

After analyzing the evidence in the literature that calcium is somehow involved in the production of cataract, the results of the experimental work of the authors are detailed, and the conclusion is arrived at that the weight of evidence seems to be in favor of the view that cataract, whether experimentally produced by naphthalene or occurring in human beings, is not the result of a lowering of the blood calcium or a disturbance in calcium metabolism. But the possibility still remains that an excess of calcium may be able to protect the lens against toxic substances, whether foreign, as in the case of naphthalene, or produced, as in the case of man, by some pathologic process. A bibliography is appended. W. ZENTMAYER.

PARATHYROID CATARACT. P. WEINSTEIN, Brit. J. Ophth. **17**: 236 (April) 1933.

The author has operated in two cases of parathyroid cataract. In one case the lens was considerably reduced in the sagittal direction, which Kast considers to be characteristic of parathyroid cataract. In both cases the lens gave a positive cysteine reaction (nitroprusside reaction). This reaction was also positive in five cases of zonular cataract. On the basis of these results, the assumption of Hesse-Phelps of the etiologic connection between zonular cataract and parathyroid tetany is corroborated. The rôle of calcium in cataract is very dubious. A bibliography is appended. W. ZENTMAYER.

ESTIMATION AND DISTRIBUTION OF ASCORBIC ACID (VITAMIN C) AND GLUTATHIONE IN ANIMAL TISSUES. T. W. BURCH and J. W. DANN, Nature **131**: 469, 1933.

Ever since the discovery of ascorbic acid it has been evident that when this acid occurred in the tissues, it was estimated as glutathione in the iodine titration. The tissues known to be rich in glutathione have also a high ascorbic acid content. In the sheep lens, ascorbic acid is 0.46 mg. per gram, or 32 per cent of the iodine titer. Ascorbic acid and glutathione may be two linked factors in one system of oxidation in the animal cell. J. E. LEBENSOHN.

THE CONTENT OF SULPHYDRYL COMPOUNDS AND ASCORBIC ACID IN OCULAR LENSES. H. VON EULER and C. MARTIUS, Ztschr. f. physiol. Chem. **222**: 64, 1933.

Vitamin C has been identified as ascorbic acid. Since both glutathione and ascorbic acid reduce methylthionine chloride, U. S. P. (methylene

blue), iodine and lead salts, all the earlier data on the glutathione and cysteine content of the lens must be revised. By the use of Tillman's indicator (2,6-dichlorphenol-indophenol) at p_H 2.5, the amount of ascorbic acid can be independently measured. The substraction of this value from the iodine titer gives the glutathione content. Such analyses show that about one fifth of the total reduction power of the bovine lens is derived from vitamin C.

Two clear lenses of a man of 30 years were also examined. Ascorbic acid accounted for 28 per cent of the reduction power. On the other hand, in cataractous lenses no vitamin C can be demonstrated.

J. E. LEBENSOHN.

Methods of Examination

RECENT OBSERVATIONS ON THE PROLONGED OCCLUSION TEST. F. W. MARLOW, Am. J. Ophth. **16:** 519 (June) 1933.

The author feels that the work of Abraham and Beisbarth is not conclusive. He reviews his originally reported cases and states that while in a great many cases there is a tendency for an occluded eye to turn upward there are exceptions, and the deviation is usually different on the two sides. He cites ten cases, and suggests that before prescribing glasses, occlusion of each eye should be done, and sursumduction and muscle balance in general should be considered.

W. S. REESE.

LOCALIZATION ON THE FUNDUS; CONTRIBUTIONS ON MERIDIAN FAULTS. M. KLEIN, Brit. J. Ophth. **17:** 145 (March) 1933.

A perimeter for localization on the fundus is described for which are claimed greater simplicity and possibility of direct transfer of the results obtained to the eye than for any heretofore used. The method of localization is detailed. The author gives the following summary:

1. The amount of the deviation caused by faulty meridians was determined with regard to the spot on which most Gonin operations are performed. In cases of error of 10 degrees these faults are about 2 mm., and in those of 15 degrees, 3 mm. If determination of the meridians takes place according to the face of the clock, 10 degrees is equal to one third of an hour and 15 degrees, to one half. In determinations based on estimation, such mistakes may easily occur, and in themselves may make the success of the operation doubtful.

2. The localization on the fundus can be most exactly made with the aid of a perimeter, because the meridian and the latitude are determined in smaller units, and thus the possibility of faults occurring is lessened.

3. Besides being easy to manipulate, the proposed perimeter makes it possible to project the meridian onto the eye and thus faults connected with the transfer can be avoided.

W. ZENTMAYER.

Neurology

THE DISTURBANCES IN VISION AND IN VISUAL FIELDS AFTER VENTRICULOGRAPHY. CLEMENT B. MASSON, Bull. Neurol. Inst. New York **3**: 190 (June) 1933.

Temporary blindness occurred in six of a hundred consecutive cases in which air was introduced into the ventricles after puncture of the occipital poles of the brain for roentgen examination. Three patients had normal fundi, and three had papilledema of from 2 to 4 diopters. In five patients vision was normal, or nearly so, and in one, it was reduced before ventriculography. One patient had a homonymous hemianopia, and one had contracted fields before the procedure. In three cases, there were indications of increased intracranial pressure.

In two patients, vision began to fail during the manipulations incident to the introduction of air; in the remaining four, it began to fail two, three, four and sixteen hours after the procedure. The amount of air introduced varied; some ventricles were partially and some were completely filled. A double Babinski reflex, which soon disappeared, developed in one patient. No other neurologic disturbances were noted.

In two cases, the reflex path for the contraction of the pupils to light was active during the period that vision was lost, and in one case the pupils were at first widely dilated and did not contract even to light, while some vision remained. Notes were not complete on the other three cases.

Visual acuity was depressed to perception of hand movements in two cases and was lost in the other four. In all of the cases the period of blindness lasted from twenty-one to seventy-two hours, four of the patients being operated on during the period. All of the patients eventually regained the visual acuity which they had possessed before ventriculography. It required from seven to thirty days for vision to return to normal (no definite figures are given) after the period of reduced vision.

The author feels that while the loss of vision in these cases was in some way connected with the puncture of the brain, he does not believe that the nature and situation of the cranial lesion, the introduction of air or the trauma to the occipital lobe by the actual puncture could be considered as the sole determining factor in any case.

W. F. DUGGAN.

SCOTOMATA IN MIGRAINOUS SUBJECTS. T. H. BUTLER, Brit. J. Ophth. **17**: 83 (Feb.) 1933.

Two cases are described. The first occurred in a man, aged 47. From 15 to 25 years of age he was subject to violent headaches associated with spots before the eyes, scintillating scotomas, hemianopsia and numbness in the arms. For the past twelve years he had been free from headache and other symptoms of migraine. A month before the author saw him he made some adjustments on his car with the engine running in a closed garage. He became giddy and almost blind in the right eye and experienced violent pains in the head. Vision in the right eye was 5/60, and in the left, 6/4. In the right eye the nasal edge of the optic disk was blurred and there was a delicate film on its surface; there were

sheaths on the arteries of the disk, either the result of an earlier neuritis or a persistence of embryonic structures. The visual field showed general contraction. The field for red showed a reniform contraction on the nasal side and a relative scotoma in a position on the nasal side corresponding to the normal blind spot on the temporal side. The medical diagnosis was migraine and carbon monoxide poisoning. The patient was seen only once.

The second case occurred in a woman, aged 45. She had suffered for years from violent headaches, especially at the menstrual periods, associated with scintillating scotomas. Nausea, vomiting and vertigo occurred. Eighteen months before the author saw her she had organized visual hallucinations. Her temper was growing bad. Each field showed slight general contraction and a hemianopic crescentic scotoma, absolute in the right and partly absolute and partly relative in the left. Both papillae showed tenuous films, and the nasal edges were blurred. There were well defined sheaths on the roots of the arteries, which appeared to be either an embryonic "rest" or the result of an earlier neuritis.

The roentgenograms were normal. Three months later the fields were normal. Papilledema was more marked on the right side.

Aneurysm and swelling of the pituitary gland were considered as possible causes of the condition. The author believes, however, that in the first case there was a vascular lesion and the association with migraine was probably fortuitous. The second case he regards as one of pure migraine.

W. ZENTMAYER.

Ocular Muscles

ORIGIN OF OCULAR TORTICOLLIS. P. SATANOWSKY, Arch. de oftal. de Buenos Aires **7:** 547 (Sept.) 1932.

The author reports a case of torticollis with inclination of the head on the left shoulder in connection with paralysis of the right superior oblique muscle. The left eye was blind. After discussing Bielschowsky's and Adrogué's explanations, the author attributes the inclination of the head: (1) to the desire to avoid diplopia; (2) to favoring of the action of the inferior rectus muscle in downward vision and to elimination of the action of the superior oblique in direct vision, and (3) to an attempt to avoid the rotation of the image. In the case reported only the last two factors came into play.

C. E. FINLAY.

Operations

RETROBULBAR INJECTIONS AND SEVENTH NERVE BLOCK. K. K. NAYAR and G. J. N. NAYUDU, Brit. J. Ophth. **17:** 235 (April) 1933.

The authors employ the O'Brien technic for blocking of the seventh nerve, and a modification of the method described by Large (*Brit. J. Ophth.* **16:** 166, 1932) for retrobulbar anesthesia of the iris. They encountered retrobulbar hemorrhage only rarely, but despite the utmost care paralysis or paresis of the inferior rectus frequently occurred. Vomiting did not follow retrobulbar injections.

W. ZENTMAYER.

Orbit, Eyeball and Accessory Sinuses

DOES TRAUMA FAVOR THE OCCURRENCE OF THE ECHINOCOCCUS IN THE ORBIT? V. P. PATEL, Brit. J. Ophth. **17**: 40 (Jan.) 1933.

In a Mohammedan boy aged 12 years, following a kick by a donkey, an increasing proptosis, upward and inward, resulted from a swelling, the size of a small orange, in the region of the right lower lid and cheek. A clinical diagnosis of an intra-orbital cyst was made, and on removal it was found to be a uniocular hydatid cyst. A similar case has been reported by Dudinow. The article is illustrated.

W. ZENTMAYER.

THE TREATMENT OF ORBITAL VARICOCELE. THE EFFECTS OF RADIUM AND SCLEROSING INJECTIONS COMPARED. L. WEEKERS, Arch. d'opht. **50**: 369 (June) 1933.

This condition is very rare. Delord and Viallefont have recently collected sixty-nine cases of intermittent exophthalmos from the literature, among which there are several with orbital varices. The differential diagnosis is sometimes difficult. In the observations collected by Delord and Viallefont nothing was said about treatment. The only cure which has been published is that of Raverdino, who gave his patient two injections of quinine hydrochlorate. Weekers describes in detail a case in which he employed radium without benefit and then obtained a cure with one injection of a solution composed of quinine hydrochlorate 2 Gm., ethyl carbonate (urethane) 1 Gm. and distilled water 15 cc. He briefly reviews the origin of the treatment of variocele by injection and mentions the various agents employed. The comparative study of the effects of radium and sclerosing injections which this personal observation afforded leads him to believe that injection of sclerosing agents is the procedure of choice in varicocele of the orbit. S. B. MARLOW.

PULSATING EXOPHTHALMOS. LIGATURE OF THE INTERNAL CAROTID ARTERY. PERRIN and WERTHEIMER, Arch. d'opht. **50**: 395 (June) 1933.

A case is reported in detail because of a very satisfactory operative result which has been maintained and has tended to improve. The patient sustained an injury to the head in a motorcycle accident three years prior to observation. Compression of the carotid was tried before operation, and this completely relieved the subjective symptoms. Subsequently the internal carotid artery was tied, after appropriate preliminary treatment. Twenty days later the exophthalmos was reduced one half and subsequently further improvement occurred.

S. B. MARLOW.

THE PATHOGENESIS AND CLINICAL PICTURE OF POLYSINUSITIS WITH ORBITAL COMPLICATIONS. G. RUSSO-FRATTASI, Ann. di ottal. e clin. ocul. **61**: 187 (March) 1933.

Four cases are reported. In the first the frontal and ethmoid sinuses were involved in a purulent infection. An opening in the floor of the frontal sinus communicated with the infected orbit. Death from men-

ingitis occurred eight days after operation. In the other three cases the orbit was apparently infected from the maxillary sinus, but the ethmoid sinuses were also affected and were possibly the primary source of infection. The ethmoid cells are considered the most commonly affected of all the sinuses and are perhaps the most common source of orbital complications. S. R. GIFFORD.

Pharmacology

KARBAMINOYLCHOLIN, A NEW MIOTIC: PRELIMINARY REPORT. K. VELHAGEN, Klin. Monatsbl. f. Augenh. **90:** 512 (April) 1933.

Karbaminoylcholine, a choline ester, is a strong miotic. A solution of 1:4,000,000,000, corresponding to the dilution of a hormone, still irritates the isolated sphincter of the pupil. It causes active miosis when instilled into the conjunctival sac, and a 0.75 per cent solution is equivalent to a 2 per cent solution of pilocarpine in glaucoma. Adverse effects have not been observed so far, but insufficient data prohibit an exact idea at the present time as to the effect of the use of this preparation for many months. A decided chemical stability increases its action. K. L. STOLL.

The Pupil

PUPILLARY TEST FOR DIAGNOSIS OF PREGNANCY. C. BERCOVITZ, Am. J. Obst. & Gynec. **25:** 882 (June) 1933.

Bercovitz studied the reaction of pregnant women, nonpregnant women, and men. One drop of a 10 per cent solution of sodium citrate is mixed with 5 or 6 drops of the patient's blood and instilled into one eye. The other eye is used for control observation and comparison. The test requires about two minutes and the reaction usually lasts about five minutes. Of the 138 women who were not pregnant none showed a positive pupillary reaction. Sixteen men gave negative reactions. There were no false positive reactions. In 183 patients the diagnosis of pregnancy was confirmed by subsequent events. Of these, 155, or 84.7 per cent, showed positive pupillary reactions. In 1 there was a positive pupillary reaction twelve days after the onset of the last menstrual period. Forty-one women were observed post partum. Thirty-four gave negative pupillary reactions. One who had a positive reaction before delivery gave a negative reaction fifteen minutes after the delivery of twins and the placenta. Phenol, 0.2 per cent, in physiologic solution of sodium chloride will prevent or abolish a pupillary reaction of pregnancy. Neutralized female sex hormone, isolated from the urine of pregnant women, failed to cause a pupillary reaction when instilled into the conjunctival sac of 14 pregnant women. All these women reacted positively to their own blood. [J. A. M. A.]

Physiology

THE PHYSIOLOGICAL SIGNIFICANCE OF WEBER'S LAW AND COLOUR CONTRAST IN VISION. J. S. HALDANE, J. Physiol. **79:** 121, 1933.

The author describes a number of psychophysiologic experiments which show that Weber's law does not hold except under exceptional

conditions of illumination. The article does not lend itself to abstracting. The author's summary is as follows:

"1. Weber's law, as applied to vision, only holds under exceptional conditions: we are ordinarily aware of arithmetical, and not geometrical, differences in either intensity or tint of illumination.

"2. Coloured shadows and similar contrast phenomena do not represent what we are accustomed to call the natural tints of either of the two surfaces concerned, but exaggerated or distorted differences in these tints.

"3. The phenomena represented in Weber's law, in ordinary vision of differences in illumination and tint, and in colour contrast, are summed up in the physiological conclusion that the field of visual activity, regarded as a whole, tends to exhibit constancy with respect to the prevalence within it of brightness and darkness, and of pairs of complementary colours, the latter being regarded as together equivalent to a degree of whiteness.

"4. Young's three-colour theory of colour vision does not cover the phenomena of colour contrast."

F. H. ADLER.

GLUTATHIONE IN OPHTHALMIC DISEASES. M. VELE, Ann. di ottal. e clin. ocul. **61:** 170 (March) 1933.

Vele estimated the glutathione in the blood of 50 normal persons and in that of 148 patients with various ocular diseases. In the normal persons the average glutathione content was 18 mg. per hundred cubic centimeters, while all the patients with ocular diseases showed higher amounts, varying from 37 mg. in corneal diseases to 31.02 mg. in senile cataract. There was also an increase in the ratio of glutathione to the red blood count. The significance of these findings is not made clear except that they indicate increased tissue metabolism. A bibliography is given.

S. R. GIFFORD.

THE MINERAL CONTENT OF THE EYE. F. P. FISCHER, Arch. f. Augenh. **107:** 295, 1933.

Chemical analyses were made of the mineral content of the various tissues of the eye. The chlorine, phosphorus, sulphur, sodium, potassium, calcium and magnesium content of the cornea, lens, vitreous, sclera, retina, iris, ciliary body and choroid were determined.

Each tissue was found to have a relatively constant and characteristic mineral content, differing considerably in nearly every case from that of the tissue or fluid in contact with it. From this fact the author concludes that thermodynamic equilibrium, as postulated by the Donan theory, cannot exist in the eye so long as the tissues are alive. The function and metabolism of each tissue necessitate a characteristic mineral content, which could be found on analysis.

Fischer believes that the mineral content of each tissue is kept constant within normal physiologic limits by some regulating mechanism.

FRANCIS H. ADLER.

Refraction and Accommodation

LENS REMOVAL FOR HIGH MYOPIA. R. O'CONNOR, Am. J. Ophth. **16:** 516 (June) 1933.

The author reports six cases in which the lens was removed for high myopia. Most of these were done by discission. One was extracted by the Homer Smith method under a Kuhnt bridge flap. The results were very good, several patients securing much better corrected vision than they had before operation.

W. S. REESE.

ETIOLOGY AND TREATMENT OF HIGH MYOPIA (ECTATIC SCLEROCHOROIDITIS). LEMOINE and VALOIS, Arch. d'opht. **50:** 245 (April) 1933.

The authors, who believe in the dualistic theory of myopia, present in this report some new arguments drawn from clinical observations in support of this theory. In their opinion, the most important indication that the myopia present is of the malignant type is the early diminution of visual acuity in spite of the wearing of glasses. In all patients whose vision with glasses falls below two thirds of normal the condition is of the progressive type, and on this basis they found that progressive myopia occurred in 17 per cent of their cases of myopia, a percentage which corresponds closely to that of Knies, 20, which was based on the depigmentation of the choroid. Women were affected in 77 per cent of cases. If poor vision was the result of ectasia of the posterior pole, there should be a strict parallelism between the refraction and the acuity. The authors present a table which shows that this is not the case. In some cases this can be charged to poor health, improper development and heredity. In others examinations of the patients and therapeutic results have led the authors to attribute the myopia to an infectious cause. As infectious causes they list, in the order of frequency, tuberculosis, syphilis and suppuration of the sinuses. Lacking absolute proof, they present as strong presumptive evidence the improvement of vision in forty-six of sixty-two patients treated over a long period with tuberculin. Some striking cases are reported. The improvement following antisyphilitic treatment is briefly noted, and a case in which drainage of an infection of the antrum resulted favorably is described. The relationship between the thyroid and high myopia is also discussed.

The two most constant symptoms of malignant myopia in its earliest stages are amblyopia and depigmentation. Its development varies. It may have a subacute onset with rapid loss of vision, or the course may be more chronic. The authors attribute the cause to sclerochoroiditis, which softens the sclera, allowing it to stretch. Treatment should be directed to the etiologic cause and should be instituted early. If this is done and carefully followed through, the authors believe that there is some hope that the process may be arrested. The forty-six cases in which the patients were benefited by treatment with tuberculin are presented in tabular form.

S. B. MARLOW.

EXTREME HYPEROPIA. S. PADOVANI, Arch. di ottal. **40:** 145 (April) 1933.

A case is reported of hyperopia of 21 diopters in one eye and 17 diopters in the other, combined with 1 diopter of oblique astigmatism in each eye. The fundi were normal, but the eyes were quite small, and vision was only 1/100 in each eye. S. R. GIFFORD.

Retina and Optic Nerve

OPTIC PSEUDONEURITIS AND PSEUDOPAPILLEDEMA. R. K. LAMBERT and H. WEISS, Arch. Neurol. & Psychiat. **30:** 580 (Sept.) 1933.

Lambert and Weiss follow a classification of pseudoneuritides somewhat similar to that of de Schweinitz (*Am. J. Ophth.* **11:** 985, 1928). The first group is found in the presence of marked refractive errors. In this group the changes in the nerve head resemble optic neuritis rather than papilledema and are so characteristic that they offer little diagnostic difficulty. The second group consists of conditions resembling optic neuritis, in which there are congenital changes in the disk and blood vessels, frequently with deposits of glial tissue. These cases, also, do not offer any considerable diagnostic difficulty. The third group is composed of conditions in which the nerve head resembles that found in true papilledema rather than that seen in optic neuritis. These cases offer considerable difficulty in differential diagnosis.

The authors followed a series of cases of pseudoneuritis for a number of years, keeping records of the blind spots and angioscotomas and making photographs of the fundi. They conclude that if these cases are followed closely by photographs the cases of pseudoneuritis will be differentiated from those of true neuritis or papilledema by the permanency of the picture. Further, in pseudoneuritis there is never any enlargement of the blind spot or any enlargement of the angioscotoma as outlined by Evan's technic. F. H. ADLER.

MARKED PAPILLOEDEMA IN PULMONARY EMPHYSEMA. A. J. CAMERON, Brit. J. Ophth. **17:** 167 (March) 1933.

Marked papilledema was seen in a coal miner, aged 34. There was intense venous congestion of both retinas. The veins were bulging, tortuous and plum colored, and there were a few retinal hemorrhages. The swelling of the right nerve head was 5 diopters and of the left, 6 diopters.

The diagnosis was extreme emphysema, unusual in that the cyanosis and congestion were confined to the upper part of the body.

No similar case was found in the literature. W. ZENTMAYER.

ATROPHY OF THE OPTIC NERVE TREATED WITH RETROBULBAR INJECTIONS OF ATROPINE. GERTRUDE SPRINGOVITSCH, Klin. Monatsbl. f. Augenh. **90:** 343 (March) 1933.

Fifty-one eyes of twenty-eight patients with atrophy of the optic nerve were treated with retrobular injections of atropine at the University Eye Clinic at Riga, during a period of eighteen months. Tables show the results of this treatment, which not only succeeded in stopping

the deterioration of vision but caused improvement. Any kind of optic atrophy could be influenced favorably, including atrophy due to methyl alcohol intoxication, tumors of the brain, injury or syphilis. General treatment was continued. Recurrences of failing vision required another course of injections. The number of retrobulbar injections varied from one to twenty, and the doses varied between 0.2 and 1 cc. of a 1:1,000 solution of atropine. Additional subconjunctival and subcutaneous injections of atropine were given in some instances. The injections, made alternatingly at the lateral and the medial side of the globe, were repeated two or three times a week until no further increase in vision was noticed. No complications were noticed in adults or in children 11 or 12 years of age, aside from slight edema of the upper lid, occasional small subconjunctival hemorrhages and dryness in the throat. The fundus remained unchanged.

Color perception returned; the field of vision became larger in some cases, as illustrated by charts, and vision was completely regained and scotomas disappeared in a number of patients. K. L. STOLL.

DIFFICULTIES IN LOCATING RUPTURES OF THE RETINA. H. ARRUGA, Klin. Monatsbl. f. Augenh. **90:** 753 (June) 1933.

The author recommends a strong light of from 200 to 400 candlepower for examination of the entire fundus by the inverted method, which allows control of the periphery even in eyes with a hazy vitreous. Exact examination of the rupture and differentiation from hemorrhage are facilitated by the direct method. The main difficulties in finding ruptures in the retina consist in their smallness, opaque media, confusion with retinal hemorrhages and location of the ruptures between folds of the retina or in the extreme periphery. Fifteen colored drawings illustrate these groups. K. L. STOLL.

FURTHER EXPERIENCES WITH THE "METHOD OF UNDERMINING" IN DETACHMENT OF THE RETINA WITH TEARS AT THE POSTERIOR POLE OF THE GLOBE: REPORT OF CASES. K. LINDNER, Klin. Monatsbl. f. Augenh. **90:** 757 (June) 1933.

The author reports his experiences with the undermining method in detachment of the retina with tears of the retina at the posterior pole of the globe. The material consists of five recent cases and four published reports (*Arch. f. Ophth.* **128:** 654, 1931). Three of the four patients are permanently cured; the fourth suffered a recurrence which resisted further treatment.

The presence of the holes was established beyond doubt after the method of Zamenhof, the only reliable one for the purpose, in the author's opinion. Aside from the retinal hole in the macula, peripheral retinal defects or traction in the vitreous existed. Lindner prefers to approach the hole in the macula from the temporal side and above the insertion of the inferior oblique, whenever possible. Insertion of the cannula and demarcation by cauterization are done as previously described, but a 4 per cent solution of potassium hydroxide, U. S. P., and a trephine 2 mm. in diameter are now used.

The author was surprised to obtain good visual results, although the choroid underlying the macula had been touched by the caustic. The result depends less on the operative method than on the location of the tear; it is less satisfactory in foveal than in parafoveal tears. Omitting to cauterize the macular tears in the presence of peripheral tears predisposes to recurrences. In fact, macular tears have been observed after an operation for a peripheral flap-shaped tear had failed. The age of the patient, the duration of the detachment prior to the operation, the number and type of the operative procedures and the final results are noted in a table.

Lindner considers this operation superior to other methods in detachment of the retina at the posterior pole of the globe. K. L. STOLL.

THE OPERATION FOR DETACHMENT OF THE RETINA WITH POTASSIUM HYDROXIDE AND ITS PROSPECTS. GUSTAV GUIST, Klin. Monatsbl. f. Augenh. **90**: 771 (June) 1933.

The author renders a detailed description of his operative method for detachment of the retina by cauterization with potassium hydroxide, U. S. P. The pathologic condition of the retina is described and illustrated by nineteen photomicrographs and photographs of the fundus. The shape and localization of the tears, the influence of the condition of the vitreous on the detachment of the retina, the proper time for the operation and the types of the detachments from a clinical-ophthalmoscopic and an operative standpoint are thoroughly discussed. The operative technic is laid down for various types of detachments, caused by cystic holes, in the upper and lower hemispheres of the globe, for closure of holes in the macula, for large flap-shaped tears and for operations devised to make the globe smaller. Regulations for the after-care are given and the complications enumerated. Besides infections, necroses and hemorrhages, lesions by faulty nutrition of the eye occur, especially after resection of several muscles, followed by destruction of the cornea and atrophy of the globe.

It is difficult to make a prognosis, but it may be said that in detachments of less than six months' duration there are good visual results. The field of vision for white returns immediately; it is slower for red, and slowest for blue, which is confused with green at first. The micropsia following the operation with potassium hydroxide and lasting from six to eight months in some cases is unexplained. Guist obtained 85 per cent of cures with his method. K. L. STOLL.

FURTHER OBSERVATIONS ON THE OPERATIONS FOR DETACHMENT OF THE RETINA AFTER THE METHODS OF GUIST AND WEVE. MARTIN BARTELS, Klin. Monatsbl. f. Augenh. **90**: 800 (June) 1933.

Sixty per cent of favorable results obtained by divers operative methods for detachment of the retina at the hands of Gonin, Weve, the author and other operators prompt Bartels to express the opinion that it is not the question nowadays whether an operation should be performed in detachment, but which method should be used. The best method is the one with which the operator is most familiar. Bartels prefers the Guist method, and he gives statistics of the various types of detachments, duration, age of the patients and operative results.

These statistics show that the results of the operation after Guist's method depend on whether the detachment is total or not, and whether a distinct tear can be found. No cure was obtained without complete closure of the tear. Most favorable are the tears in the temporal upper portion of the retina. The results were more favorable in men than in women, and less satisfactory the older the patients were, especially in those over 50 years of age. The condition of the vitreous seemed to be irrelevant. A certain number of trephining holes is necessary, but too many, e. g., from fifteen to nineteen, are unfavorable. Hemorrhages did not interfere with the result, except in one case. Secondary tears were not observed.

Bartels operated in seven cases with electrocoagulation after the method of Weve with doubtful results. Weve's method seems to be indicated in tears of the ora serrata, which, however, healed well also after Guist's method, although it is harder on the patient and the operator. General constitutional elements, age and sex of the patient and the type of the detachment play a decided rôle in the use of Guist's method. Operation should be performed in every case, including the unfavorable ones, in an attempt to gather further experience which will help to obtain cures in the remaining 40 per cent of failures.

After giving due credit to Gonin as the originator of the retinal operation, Bartels again points out the necessary closure of the tears and refers to Lobeck's experiments which showed the difference of the current in the posterior portion of the globe under normal conditions and after the development of a tear in detachment of the retina.

K. L. STOLL.

Trachoma

EXPERIMENTAL RESEARCH ON TRACHOMA. P. THYGESON, Arch. Inst. Pasteur de Tunis **22**: 157, 1933.

Bacillus granulosis was not isolated in sixty-three patients studied. In 46 per cent a morphologically similar bacillus was encountered, and in 20 per cent, a bacillus of the Koch-Weeks type. These must be considered as accidental invaders, as neither was capable of producing trachoma on the human conjunctiva.

J. E. LEBENSOHN.

CONTAGIOUSNESS OF TRACHOMA: THREE NEW CASES OF DIRECT CONTAGION. CUENOD and NATOF, Arch. d'opht. **50**: 261 (April) 1933.

The cases of three physicians, all of whom were infected accidentally during operations on trachomatous patients, are described. Two received immediate treatment, consisting of lavage and the instillation of silver nitrate, only to have the disease become manifest later. These cases happened under perfect scientific conditions, so that there can no longer be any doubt as to the contagiousness of trachoma. The question of microbiotropism and the relationship of Bacillus granulosis to it are discussed.

S. B. MARLOW.

Tumors

LACRIMAL ADENOMA. H. VILLARD and C. DEJEAN, Arch. d'opht. **50:** 348 (May) 1933.

Although about twenty cases have been reported in the literature, only four or five can be accepted unconditionally. This number should be still further reduced if it is insisted that adenoma, a proliferation of the gland in all its aspects, should also function physiologically as evidenced by swelling of the gland during lacrimation. The authors present a clinical and detailed histologic description of a personal case conforming to the latter criterion. They emphasize especially the curious symptom of swelling of the gland under the influence of crying.

S. B. MARLOW.

INTRASCLERAL EPITHELIAL CYST. D. MICHAIL, Arch. d'opht. **50:** 385 (June) 1933.

Cysts of this type are very uncommon and usually follow a perforating injury of the globe or, exceptionally, an operation. The author reports a personal observation including complete histologic study. His case concerned a young man, 24 years old, who had received a large perforating wound at the limbus with expulsion of the lens and prolapse of the ciliary body. The cyst appeared three years after the accident. The cyst, which had invaded the whole atrophied globe, was traversed by a scleral band containing a blood vessel. It was covered by a layer of stratified epithelium resembling corneal epithelium. This epithelial lining projected finger-like elongations into the region of the old injury. The author lists other similar cases which have been reported. A point which is brought out by the study of these reports is that the reactions and painful symptoms which may develop are always indicative of the great probability of extension within the eye. S. B. MARLOW.

Society Transactions

EDITED BY DR. JOHN HERBERT WAITE

NEW YORK ACADEMY OF MEDICINE, SECTION OF OPHTHALMOLOGY

GUERNSEY FREY, M.D., *Secretary*

Nov. 20, 1933

MARK J. SCHOENBERG, M.D., *Presiding*

VIABILITY OF THE MARGIN OF THE LID WITH CILIA AFTER TWENTY-FOUR HOURS IN THE EYEBALL. DR. BEN WITT KEY.

This article was published in full in the April 1934 issue of the ARCHIVES, page 687.

ORBITAL LYMPHOMA IN CHRONIC LYMPHATIC LEUKEMIA. DR. MARTIN COHEN.

This article was published in full, with the discussion, in the April 1934 issue of the ARCHIVES, page 617.

REMOVAL OF A TUMOR OF THE ORBIT, WITH NEURECTOMY OF THE OPTIC NERVE. DR. THOMAS H. JOHNSON.

A woman, aged 32, was presented, who for one and one-half years had shown increasing exophthalmos of the right eye, measuring at the time of the report 28 mm. by exophthalmometer. Except for an enlarged right blind spot the visual fields were normal, and vision was 20/30. Roentgen examination disclosed no abnormalities, except that the right optic canal measured 5.5 and the left 4.5 mm. The results of a complete general physical examination were negative.

The orbit was explored through an external canthotomy, and a tumor (33 by 24 by 18 mm.) surrounding the optic nerve was removed, along with that portion of the optic nerve included in the tumor. The pathologist reported that a normal optic nerve extended entirely through the growth, and that the subdural space was completely filled with tumor cells arising from the meninges around the optic nerve. The pathologic diagnosis was meningioma.

The fundus was observed immediately after the neurectomy, the head of the nerve and the retina were of good color, and the retinal vessels were full of blood. At the expiration of a week no changes could be noted in the retina. The second week after the neurectomy retinal pigment began to migrate, and at the fourth week the head of the nerve was covered with a deposit of black pigment. Many of the larger retinal arteries are still filled with blood, but the pigmentary changes seem to be progressive.

DISCUSSION

DR. H. H. TYSON: The successful removal of the tumor by Dr. Johnson again demonstrates the feasibility of the removal of orbital growths by this method. An encapsulated hemangioma of the orbit of twelve years' duration was removed recently by this method; the eye

is now practically straight, there is good motility, and the patient has normal vision.

Dr. Martin Cohen: Might it not have been possible in this case to remove the extradural growth by a Krönlein operation without interfering with the optic nerve? If the optic foramen is enlarged, the question arises whether there is not intracranial involvement.

Dr. Sigmund Agatston: It is difficult to understand how the fundus could remain normal after section of the optic nerve through the retinal artery. The retina now presents the picture of secondary retinitis pigmentosa, suggesting the possibility of some obstruction to the circulation of the retina being the actual cause of retinitis pigmentosa.

Dr. Percy Fridenberg: It would be interesting to know whether pressure on the globe could elicit retinal arterial pulsation. The secondary retinal changes are mainly degenerative, and the pigment, mainly on the disk and representing blood coloring matter, has migrated forward in the nerve or nerve sheaths from the severed vessels at the point where the nerve was cut through. All this is quite different from any picture of retinitis pigmentosa.

Dr. Thomas H. Johnson: This was not an extradural but an intradural tumor. The nerve has been sectioned, and the endothelial growth surrounds the nerve in close apposition to it inside of the dura. Cells of the type found in this tumor have their origin in the arachnoid. As to the circulation of the retinal vessels, undoubtedly there was a collateral circulation through the circle of Zinn.

Rupture of the Globe. Dr. Joseph S. Hory.

A man, aged 27, was struck by a fragment of iron chisel, which caused a complete perforation of the left upper lid and of the eyeball. Vitreous humor exuded through the rupture of the globe. The roentgen rays disclosed a foreign body (8 by 7 by 3 mm.). This was easily removed through the wound of entrance by the giant magnet. The wounds were repaired surgically, the sclera being closed with three buried silk sutures, and supported by a conjunctival flap. The healing was uneventful. Ten weeks after the accident vision was 20/40, and there have been no signs of irritation in either eye.

DISCUSSION

Dr. Mark J. Schoenberg: The interesting feature in this case is that one sees an eye with sutures passing through the sclera and choroid and involving the retina. What is the future of such an eye? Will a detachment of the retina develop, or will the eye remain quiet?

Dr. Thomas H. Johnson: I should remove the sutures. It would be a simple matter to turn back the conjunctival flap and remove the sutures. If one waits until a detachment has occurred, it will be too late to take the sutures out; the same statement applies to infection. There is also the possibility of a cyst forming around them.

Dr. E. S. Sherman: On two or three occasions I have left very fine silk in a scleral wound, covered it up with conjunctiva, and observed no ill results or bad reactions. I think that Dr. Lancaster of Boston several months ago showed that silk in the sclera is well tolerated and is best left alone.

DR. HORY: I have had only one other experience in suturing the sclera through and through. Within a period of one month infection set in and the eye was lost. I believe that in this case I should rather leave well enough alone, as four months have now passed and no irritation is present.

VISUAL RESULTS IN CASES OF INTRA-OCULAR FOREIGN BODY. DR. WALTER F. DUGGAN.

This article was published in the December, 1933, issue of the ARCHIVES, page 768.

DISCUSSION

DR. E. S. SHERMAN: The two most important factors causing loss of vision in cases of intra-ocular foreign bodies are the size of the missile and the length of time which elapses before it is removed. The size of the foreign body and its location determine whether it is to be removed by the anterior or by the posterior route. It is safer to remove a large foreign body through an incision in the sclera. If the lens is injured, there should be less hesitation about using the anterior route. If the foreign body goes through the ciliary body and is in the vitreous, it is usually better to remove it by making another incision back of the limbus. I have seen only one case of detachment of the retina following the removal of a foreign body through the sclera. This detachment was small, and reattachment followed two weeks' rest in bed.

I believe that a foreign body remaining in contact with the retina soon becomes attached by fibrinous exudate, and this attachment, rather than the removal route, constitutes the hazard in removal.

In seventy consecutive cases of intra-ocular foreign body, the point of entrance was in the cornea in thirty-four, in the sclera in twenty-eight and in the limbus in eight. There were forty-five cases of intra-ocular foreign body in the vitreous and retina, six in the lens, eleven in the anterior chamber and iris, one in the posterior chamber, two in the posterior scleral wall and five in the orbit (double perforation). Fourteen of the cases of vitreous and retinal injury resulted in blindness or enucleation. In eleven cases vision was less than 20/200; in three it was from 20/70 to 20/50; in two, it was 20/40; in six, 20/30, and in nine, 20/20. There were eleven cases of foreign body in the anterior chamber, in one of which vision was 20/30, and in ten, 20/20. The patient with foreign body in the posterior chamber had 20/50 vision, and the two patients with foreign body in the posterior scleral wall, less than 20/200. Of those with double perforations, two had less than 20/200 vision, one had 20/100, one had from 20/50 to 20/70 and one had 20/20. The patient with 20/20 vision had a small spicule of steel which entered through the sclera near the limbus, went through the fundus and was seen in the orbit. There was very little reaction. He was followed for nearly two years, and there was no intra-ocular disturbance.

Of the forty-five cases of foreign body in the vitreous or retina, removal was accomplished by the anterior route in seventeen and by an incision in the sclera or through the original scleral wound in twenty-four. Four foreign bodies were not removed for various reasons. Of the seventeen cases in which the foreign body was removed by the

anterior route, four resulted in blindness or enucleation; in five vision was less than 20/200; in one it was 20/40; in four, 20/30, and in three, 20/20.

Of the twenty-four cases of removal through the sclera, seven resulted in blindness or enucleation; in five vision was less than 20/200; in three it was from 20/50 to 20/70; in one, 20/40; in two, 20/30, and in six, 20/20. In this series, there were four cases of siderosis.

Dr. George S. Dixon: In the early days we found quite a number of old cases in which a foreign body had remained in the eye for five years or more. I distinctly remember one case in which it had remained for forty-two years.

It is remarkable that many eyes with retained foreign bodies will remain free from symptoms for so long. During the war, if a foreign body could not be removed at once and the eye did not show alarming symptoms it was left alone. Many eyes contained small pieces of shrapnel.

Dr. Clyde E. McDannald: I should like to ask Dr. Duggan how many cases of siderosis he had, and whether or not any of them cleared up.

A patient consulted me in December, 1917, with a pronounced siderosis and a cataractous lens of the left eye. Roentgenographic examination showed a small foreign body, 10 mm. back in the vitreous. Two attempts were made to remove it with the giant magnet; both were unsuccessful. On Jan. 21, 1918, the lens was removed by linear extraction. The visual result obtained was 20/30, corrected. I did not see this patient again until 1932, at which time the siderosis had completely disappeared.

It seems to me that it is becoming increasingly more difficult to remove foreign bodies by means of a magnet. In discussing the subject with an engineer, he suggested that this may be due to the fact that nonmagnetic substances are added to steel for the purpose of increasing the efficiency of the metal for certain uses.

Dr. Joseph S. Somberg: I believe that one of the most interesting questions in regard to intra-ocular foreign bodies is: "How early does siderosis develop?" I have never seen siderosis develop so as to make a diagnosis possible until six months after the entrance of the foreign body.

In the past few years I have been applying the electrocautery to the wound in the sclera after the removal of foreign bodies by the posterior route and have encountered no late complications such as retinal detachment. In other words, it seems desirable to create an adhesive process which prevents retinal detachment.

Dr. Morris Davidson: I should like to ask Dr. Duggan and Dr. Sherman about their observations in regard to sympathetic ophthalmia following intra-ocular foreign bodies.

Recently I reviewed seventeen cases, and the earliest appearance of siderosis was in two and one-half months. The interval depends on the method of examination. The fine subcapsular deposits in the lens are under the iris and not in the pupillary zone, and they are not discovered early, unless one examines these eyes carefully in maximal mydriasis with a slit lamp.

DR. DAVID H. WEBSTER: The earliest case of siderosis I ever saw occurred one week after the injury. This year we had a case at the Manhattan Eye, Ear and Throat Hospital in which the eye, showing siderosis microscopically, was removed the day following the injury.

DR. JOSEPH SOMBERG: I should like to ask Dr. Webster whether there had been any previous injury to that eye and whether he thinks that siderosis is simply an oxidation. The siderosis I am discussing is of the type which develops from a retained intra-ocular foreign body until a very slow process of oxidation occurs with gradual circulation of the oxidation process through the tissues of the globe with subsequent retention in various cells. How does Dr. Webster think that such a process could occur within one day?

DR. DAVID H. WEBSTER: I have no explanation for the fact, but I have seen oxidation outside the eye occur within a few hours. The fact that it takes place within the eye does not necessitate any definite period of time before the chemical change takes place. I think that much depends on the kind of steel which enters the eye.

DR. W. GUERNSEY FREY, JR.: The development of a ring of rust about a corneal foreign body is a comparable chemical reaction, and may take place within a few hours.

DR. E. S. SHERMAN: The shortest time which I have known to elapse between an injury and the recognition of siderosis was five months. It is likely that considerable change has gone on in many cases before one recognizes the condition as siderosis. There are probably many cases which, if examined closely, would show evidence of siderosis as early as two or three months. There are records of well authenticated cases having appeared in three months.

DR. JOSEPH LEVINE: I believe that it would be hard to recognize siderosis in early cases unless it occurred under the lens capsule. I demonstrated it pathologically after one day in Dr. Webster's case. I have looked up the literature in this particular condition, and can state that another case is reported in which siderosis was seen microscopically one day after the foreign body entered the eye; in two cases it was seen in three days.

Dr. Somberg's question as to why this can occur so quickly has been answered by others who have studied the subject, and they all think that it depends on the type of iron or steel and on its situation in the globe. If the foreign body happens to be situated in the lens no siderosis will occur, because this area is avascular. If it is situated in a part of the eye which is vascular, siderosis will occur, and if it is situated in a part of the eye which is very vascular, siderosis will occur quickly. Lloyd reported a case in which the foreign body was situated in the anterior chamber, and no siderosis occurred.

DR. JAMES W. SMITH: I should like to inquire of Dr. Duggan as to the opinion of the ophthalmologists whom he interviewed regarding sympathetic ophthalmia, and the "danger zone" in the ciliary region. I should be apprehensive regarding the possibility of sympathetic ophthalmia in any case in which a large foreign body entered the ciliary body even after the foreign body has been removed.

DR. PERCY FRIDENBERG: There are clinical siderosis, microscopic siderosis and subjective siderosis. How can one tell when siderosis

began? The fact that one finds a slight trace of it does not mean that it began an hour or a week before. A change in the color of the iris indicates that siderosis has been present for some time. Possibly if we could examine a patient with a slit lamp every day after the removal of the foreign body, we could find the siderosis very early, but patients come when symptoms appear, and that may mean months after the inception of the condition.

DR. MORRIS DAVIDSON: I have observed, but I cannot substantiate the observation by a sufficient number of cases, that in cases in which there has been a perforation of the zonule by the foreign body, as disclosed by an accompanying iridodialysis, siderosis has shown up earlier than when no such perforation was present.

DR. WALTER F. DUGGAN: Unfortunately, the majority of the sixteen cases of siderosis were not seen at the Herman Knapp Memorial Eye Hospital until from two months to twenty years after the injury, so that the siderosis had developed before they came to us. There was no record of the siderosis clearing up in any of the cases.

The majority of cases of siderosis resulted from steel in the choroid, vitreous or ciliary body. The percentage of incidence was 26.6 per cent for the choroid, 17.6 per cent for the ciliary body and only 4.7 per cent for the vitreous.

Dr. Smith asked about the danger zone of the eye. In my total series, when the foreign body was in the ciliary body or the vitreous, enucleation was done in 29.4 per cent of the cases; when the foreign body was in the choroid, in 40 per cent, and following a double perforation, in 42.8 per cent.

I believe that Dr. Davidson asked about sympathetic ophthalmia. There were only two cases in the whole series; both of these are described in my report.

Dr. Sherman's discussion was interesting, and I noticed that his statistics roughly paralleled mine. One interesting fact is that the patients followed up for one year or more showed slightly better visual results than those followed for less than a year. The patients followed for two years or more had vision about midway between that of the patients followed for one year and that of the patients followed for less than one year. It may be that in my series the patients who had good vision stopped coming back after a year, while those whose vision was not as good kept coming back with the hope that something might be done for them.

ROYAL SOCIETY OF MEDICINE, LONDON, SECTION ON OPHTHALMOLOGY

Jan. 12, 1934

A. C. HUDSON, F.R.C.S., *President*

THE SPREAD OF THE IRON COMPOUND IN SIDEROSIS BULBI. MR. EUGENE WOLFF, F.R.C.S.

In reading accounts of siderosis bulbi, it is difficult to understand why certain portions of the eye take up the rustlike substance, while others

do not. It seems unnecessary to ascribe a special affinity to certain tissues, except that, in all probability, connective tissues take up iron less readily than do other tissues.

The march of events in siderosis bulbi is quite definite, as can be shown by taking it in its three stages. In the first, the latent period, iron is converted into a diffusible compound, which stains the surrounding tissues, and which makes its way into the aqueous and vitreous. The theories are: 1. Iron may be dissolved by the carbon acid of the tissues, or by the acid phosphates. 2. It may enter into solution in organic form as an albuminate or as a colloid. In siderosis bulbi, the vitreous has often been found rust-colored.

The second stage is that of the spread of iron derivatives through the ocular fluids. In this process the iron stains all the structures with which it comes in contact. The epithelium of the lens takes up the compound very readily, particularly the proliferating cells, which accounts for the characteristic brown patches seen in the lens. The ligamentum pectinatum iridis is frequently stained. The iron compound diffuses through the cornea, staining the substantia propria, the corneal corpuscles and, eventually, the corneal epithelium. That the retina is affected from the side of the vitreous was strikingly shown in a case described by Morax, in which the ganglion cell layer was affected, but the remainder of the retina was scarcely involved.

The third stage is characterized by degeneration in the retina and the lens. The ganglion cells and other nervous elements of the retina degenerate, and neuroglia cells proliferate. The retinal pigment epithelium proliferates, becomes mobile and invades the retina, and pigment tends to collect around the vessels. Finally, the retina may become detached. The lens becomes opaque. It is possible that changes in the ciliary body may cause a more albuminous dialysate and produce the rise of tension seen in some cases.

DISCUSSION

MR. A. C. HUDSON: I wonder how many members have seen siderosis of the retina clinically. Most of the patients get cataract. I remember seeing only one with retinal siderosis, and in that one the color was brown.

MR. F. A. JULER: Mr. Mayou published a paper some time ago in which he said siderosis bulbi might be due to diffusible iron; that seems to be the same theory which Mr. Wolff is now advancing.

MR. EUGENE WOLFF: The reference in Mr. Mayou's paper is to colloid, and colloid cannot get through a semipermeable membrane like the capsule of the lens. Mr. Mayou said that the iron could not get under the capsule unless there was rupture.

THREE GROUPS OF UNUSUAL DISTURBANCES OF THE FUNDUS. MR. ARNOLD SORSBY.

The first group shows a familial occurrence of macular coloboma associated with apical dystrophy of the hands and feet. Miss Ida Mann has shown that macular colobomas can be divided into three varieties:

pigmented, nonpigmented, and those associated with blood vessels. The second of these is said to be commonest, but I do not agree. Some unilateral colobomas of the macula are of inflammatory origin, but this cannot be said of all; most authorities regard them as embryologic defects. Miss Mann urges the view that colobomas are of inflammatory origin, some from postnatal and some from intra-uterine inflammation. Her contention is that the pigmented colobomas represent irritative lesions, and the nonpigmented ones, destructive lesions. The theory of an inflammatory origin is seriously challenged by three family groups which have been reported by Clausen, Schott and Davenport. Clausen reported the presence of bilateral coloboma of the macula in a brother and a sister. Two sons of the brother and two daughters of the sister were likewise affected. Schott reported the condition in two daughters of an unaffected man whose sister was affected. Davenport reported the lesion in both a mother and her son.

The cases shown tonight are examples of the first group. The mother has bilateral coloboma of the macula. Of seven children, five show the same defect. The lesions are of the pigmented variety. The parents are not consanguineous, and no macular coloboma could be traced in any members of the mother's family. In both the mother and the affected children, the Wassermann reaction was negative. An important feature in this group is the associated skeletal defect. There are absence of nails, especially on the index fingers and big toes, stunting of phalanges and a tendency to bifurcation of the terminal phalanges of the thumbs and big toes. There is no such skeletal defect in the two children not affected by macular coloboma. Intelligence and mentality are normal in these subjects.

The second group is that of central and paracentral tapetoretinal degeneration, exhibited tonight in six members of one family and in two isolated cases. It is familial, and it combines features found in the other groups. All the patients show a central lesion, not confined to the macula, but without impairment of central vision. Some of the patients show optic atrophy, and in these the condition is the most advanced.

The third group is that showing cavernous atrophy of the disks associated with retinal arteriosclerosis, but not always with sclerosis of the vessels of the choroid. Characteristic optic atrophy with cupping is the dominant feature. The tension is normal at first, and the fields show uniformly a shrinking which is not glaucomatous. In seven of twenty such eyes observed, the ultimate stage has been glaucoma.

DISCUSSION

MISS IDA MANN: The reader of the paper has demonstrated a clearcut clinical condition, and the group now freshly described should receive a name. The association of fundus disturbance with deformities of the extremities has not, so far as I am aware, been pointed out previously. That colobomas are developmental in origin I do not think can be doubted; the fault must lie in the germ plasm. The failure in the eye is also, I think, entirely mesoblastic; it is a failure of choroid, not of retina. It looks as if there may be separate genetic factors for the

development of different parts of the choroid, and that there may be some association between that and the other mesoblastic elements in the body.

DR. E. A. COCKAYNE: I do not think it is necessary to try to connect causally the ocular with the skeletal defect; I do not see why this should not be considered as another example of linkage of two unrelated defects. It is likely that one of the parents, instead of having one mutation in germ cells, had two mutations and two independent genes in the same chromosome, and that her offspring had both defects, and that both defects are bound to be transferred together to every affected member. I think the associated skeletal defect must be very rare. Defects of hands and feet such as these patients present have never been properly classified or named, and therefore are not indexed for reference in the periodicals.

MR. R. AFFLECK GREEVES (showing picture): This is a picture of unilateral coloboma in a man aged 38, in whom the other eye was normal. This man had a blow in the eye with a cricket stump when he was 10 years of age, and the sight of the eye has been defective ever since.

MR. RANSOM PICKARD (after considering the question of the occurrence of apparent glaucoma without rise of tension): I have not encountered any test to separate the two kinds.

MR. A. C. HUDSON: The cases which Mr. Sorsby grouped as instances of atrophy due to arteriosclerosis are interesting. Sometimes one encounters a case of atrophy in which it is difficult to make a diagnosis, and one is worried as to whether the condition represents a mild type of glaucoma. I am reluctant to attribute the condition in such a case to arteriosclerosis.

MR. ARNOLD SORSBY: The remainder of the skeleton was roentgenographed in these cases, and no further abnormality was seen. My third group is the one I feel least confident about. I think it is a distinct group. Arteriosclerosis is always present. Cupping is not seen in the first stage, and the field is never of the glaucomatous form. Later there may be an extensive process that produces glaucoma of a chronic kind. I have seen ten cases now, and the early stages are characteristic. I wish to emphasize the peculiar field defect.

TUMOR OF THE IRIS. MR. A. F. MACCALLAN.

I presented this patient six years ago, when the lesion was thought to be a nevoid growth. He consulted me again a few days ago, not having been seen in the interval. The growth looks about as it did before. I still think it is nevoid. Seeing such a growth for the first time, one would be inclined to attribute malignancy to it.

DISCUSSION

MISS IDA MANN: I saw a woman, aged 48, with a similar condition, the tumor being on the nasal side of the iris. I am watching it, and after seeing Mr. MacCallan's patient I feel more sure that that is the right course.

Book Reviews

Clinical Studies on the Physiology of the Eye. By J. Grandson Byrne. Price, 10 shillings, sixpence. Pp. 144, with 49 illustrations. London: H. K. Lewis & Co., Ltd., 1934.

This book is in part a summary of a recent work by the same author entitled "Studies on the Physiology of the Eye," reviewed in the ARCHIVES (**10:** 721 [Nov.] 1933), and in part an application of his theory of the mechanisms governing palpebral, pupillary and lenticular movements to explain various clinical conditions.

Chapters 1 to 8 summarize his earlier work. He discusses paradoxical and pseudoparadoxical pupillary phenomena and then reports cases in which the patients had various somatic or visceral lesions in the differential diagnosis of which these pupillary phenomena were supposedly of importance. After a careful perusal of the reports, it seems to me that the pupillary phenomena contributed nothing to the diagnosis of the cases described, as the diagnosis was clearcut in every instance. Moreover, only one case of each kind is described, and there are no statistics as to the percentage of incidence of the pseudoparadoxical pupillary phenomena, so that one cannot determine whether these phenomena have even a confirmatory value in the diagnosis.

Chapter 10 deals with the mechanism of the Argyll Robertson pupil. Byrne quotes earlier workers, from 1852 to 1909, who found lesions (1) between the optic tract and the constrictor center, (2) in the ciliary ganglion, (3) in the cervical spinal cord and (4) in the cervical sympathetic nerve, and then states that various types of the Argyll Robertson pupil are found, which vary in size and shape and which show paradoxical dilatation or constriction or pseudoparadoxical dilatation (in a positive or negative phase). These variations in the Argyll Robertson pupil, according to his theory, prove that lesions in (1) involve the afferent dilator and constrictor paths; in (2), the efferent constrictor paths; in (3), the afferent constrictor paths, and in (4), the efferent dilator paths. In other words, lesions almost anywhere in the body are predicated by Byrne in order to fit the Argyll Robertson pupil to the Procrustean bed of his theory.

In support of these views, he mentions that retinal illumination always elicits a preliminary dilatation of the pupil (which is at variance with the careful experiments of Gradle and his co-workers) and that there is no such thing as accommodation without convergence. As is well known, these two functions are normally closely related, but the relation is relatively elastic, so that either can be exercised separately.

Merritt and Moore, in a recent review of the subject of the Argyll Robertson pupil (*Arch. Neurol. & Psychiat.* 30: 357 [Aug.] 1933) mentioned that the typical Argyll Robertson pupil, in addition to other characteristics, shows absence of the sympathetic ciliospinal reflex. In other words, two separate pathways of innervation of the iris are injured by a single lesion. Fibers passing from the hypothalamic sympathetic center to the centers in the spinal cord arch ventrally to the

mediolateral portion of the midbrain, running for a short distance close to the light reflex fibers. A single lesion at this point (around the aqueduct of Sylvius) would affect both sets of fibers and produce the Argyll Robertson pupil. This explanation is more rational than Byrne's, as it rests on a secure anatomic and pathologic basis and does not necessitate the hypothesis of lesions in one or several diverse locations.

The author closes with a chapter on the treatment of disorders of accommodation, in which he states dogmatically that too many bifocal glasses are worn, that the constant wearing of glasses for astigmatism is a mistake and that the maintenance of efficient accommodation depends on the daily natural (?) exercise of the external ocular muscles. It is obvious that he is aware neither of the fundamentals of refraction nor of the state of accommodation in the various types of ametropia, nor has he any conception of the differences between central and peripheral vision or the function of each.

Byrne's statements sound most learned, but, when examined carefully, they turn out to be only opinions backed by an involved and complicated theory, many points of which are either false or at utter variance with the conclusions drawn by other workers in the same field.

W. F. DUGGAN.

Determination of the Sensitiveness of the Eye to Differences in the Saturation of Colours. By L. C. Martin, F. L. Warburton and W. J. Morgan. Price, 1 shilling, net. Pp. 42, with 19 charts and diagrams and 8 tables. London: His Majesty's Stationery Office, 1933.

This is the fourth report on the subject of color vision to be published by the Medical Research Council and is in direct succession to that by Dr. W. D. Wright entitled "A Redetermination of the Tri-Chromatic Mixture Data." The subject is treated largely from a theoretical standpoint.

The spectral colors are not of equal saturation, and it is doubtful whether any spectral color is completely saturated. This condition, like color itself, is a psychologic entity and cannot be measured directly.

The authors' experiments were limited to the recognition of the steps in saturation between two contiguous halves of a field of view, the total field subtending an angle of 2 degrees square, and the line of separation being horizontal. The point at which the least difference in saturation became just perceptible, the limen of purity, was investigated under various conditions. The apparatus used consisted substantially of the trichromatic colorimeter of Wright, together with means for producing additive mixtures of the spectral colors with variable quantities of white. In the main experiments, both fields were composed of a mixture of white with the same spectral color. The field of comparison was kept constant, while the field of testing was advanced by one saturation step. The field of comparison was brought to equality with the field of testing, and the next step was then determined. Complete tables of twenty-three and twenty-four steps are given. With another method the fields were composed of a mixture of two complementary colors. The experiments were carried out by a few trained observers over a long period; the results are given in great detail.

The authors state that the Young-Helmholtz theory does not give much help in regard to capacities for discrimination. Helmholtz suggested that the discrimination of color could be studied by applying Weber's law to the Young-Helmholtz theory, and Schrodinger (1920) found that the locus of the threshold from white was a closed curve. The authors found that the curve showing the least discriminate steps of purity is an ellipse.

This report, with its complete list of charts and diagrams, will be of great service to any one interested in the subject of color vision.

JOHN B. LYNCH.

A Glossary of French Medical Terms Referable to the Eye. By Ellen Kerney, A.M. Paper. Price, $1.50. Pp. 209. New York: Institute of French Studies, Inc., Columbia University, 1934.

This dictionary of French medical terms referable to the eye will be of great aid to those who are working in French ophthalmologic literature. The author states that the work is not exhaustive and aims only to present the terms used in standard works and in recent magazine articles. Even so, its value is evident, and its appearance will be greeted with relief by those who have searched in vain in the available dictionaries for a French medical term. Frequently, not only the English equivalent, but also an English translation of the meaning of the word is added, thus, "glaucome hémorrhagique, hemorrhagic glaucoma; it is a malignant but rare form; characterized by retinal apoplexy, very violent pain and exaggerated hardness of the eyeball. (Littré Dictionnaire de Médecine, ed. 22, Paris, J. B. Baillière & fils 1908)."

The book is well printed and inexpensive and should find a place in the library of every ophthalmologist who follows the foreign literature.

A. K.

Dvorine Animated Fusion Training Chart. Price, $10. Baltimore: The Animated Fusion Training Charts Co., 1934.

This set of 101 charts for use in the stereoscope is similar to the Wells series. The chief point of difference is that some of the charts used for developing dormant vision have safety movie films mounted on disks. When one of these charts is held toward the light the film is illuminated. The charts for developing macular visual function are printed on both sides so that they may be used for training either eye. All the charts are brightly colored. Those for developing fusion and stereopsis are not up to the standard of the corresponding ones in other sets. The chief advantage of these charts is that they offer a variety of designs for young children. They may be used to supplement the Wells charts.

W. F. DUGGAN.

Directory of Ophthalmologic Societies *

INTERNATIONAL

International Association for Prevention of Blindness

President: Prof. F. de Lapersonne, 217 Faubourg St., Honoré, Paris.
Secretary-General pro tem: Dr. A. Churchill, 66 Boulevard St. Michel, Paris.

International Ophthalmologic Congress

Secretary: Dr. Francisco Poyales, Olozaga 3, Madrid.

FOREIGN

British Medical Association, Section on Ophthalmology

President: Dr. Frank H. Crawley, 5 Fitzwilliam Pl., Dublin.
Place: Bournemouth, England. Time: July 25-27, 1934.

Ophthalmology Society of Bombay

President: Dr. D. D. Sathaye, 127 Girgaum Road, Bombay 4.
Secretary: Dr. H. D. Dastur, Dadar, Bombay 14.
Place: H. B. A. Free Ophthalmic Hospital, Parel, Bombay 12. Time: First Friday of every month.

Ophthalmological Society of the United Kingdom

President: Mr. A. H. H. Sinclair, 6 Charlotte Square, Edinburgh.
Secretary: Mr. Humphrey Neame, 56 Portland Place, London, W. I.

Oxford Ophthalmological Congress

Master: Dr. Cyril Walker, Bristol, England.
Secretary: Dr. C. G. Russ Wood, Hill House, Abberbury Rd., Iffley, Oxford, England.
Time: July 4-7, 1934.

Royal Society of Medicine, Section of Ophthalmology

President: Dr. A. C. Hudson, 50, Queen Anne St., W. 1, London.
Secretary: Dr. J. D. Magor Carbell, 27, Weymouth St., W. 1, London.

Société Française d'Ophthalmologie

Secretary: Dr. René Onfray, 6 avenue de la Motte Picquet, Paris 7è.

German Ophthalmological Society

Secretary: Prof. A. Wagenmann, Heidelberg.
Place: Heidelberg. Time: Aug. 6-8, 1934.

NATIONAL

American Medical Association, Section on Ophthalmology

Chairman: Dr. William C. Finnoff, 1612 Tremont St., Denver.
Secretary: Dr. Parker Heath, 1551 Woodward Ave., Detroit.

American Academy of Ophthalmology and Otolaryngology

President: Dr. J. M. Wheeler, 635 W. 165th St., New York.
President-Elect: Dr. Wells P. Eagleton, 15 Lombardy St., Newark, N. J.
Executive Secretary-Treasurer: Dr. William P. Wherry, 1500 Medical Arts Bldg., Omaha.
Place: Chicago. Time: Sept. 9-14, 1934.

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* Secretaries of societies are requested to furnish the information necessary to make this list complete and to keep it up to date.

American Ophthalmological Society

President: Dr. W. G. M. Byers, 1458 Mountain St., Montreal.
Secretary-Treasurer: Dr. J. Milton Griscom, 2213 Walnut St., Philadelphia.
Place: Lucerne, Que. Time: July 9-11, 1934.

National Society for the Prevention of Blindness

President: Mr. William Fellows Morgan.
Managing Director: Mr. Lewis H. Carris, 50 W. 50th St., New York.
Place: New York. Time: November, 1934.

SECTIONAL

New England Ophthalmological Society

President: Dr. Hugo B. C. Riemer, 128 Newbury St., Boston.
Secretary-Treasurer: Dr. Benjamin Sachs, 483 Beacon St., Boston.
Place: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston. Time: 8 p. m., third Tuesday of each month from November to April, inclusive.

Pacific Coast Oto-Ophthalmological Society

President: Dr. A. W. Morse, 66 W. Park St., Butte, Mont.
Secretary-Treasurer: Dr. Frederick C. Cordes, 384 Post St., San Francisco.
Place: Butte, Mont. Time: July 16-18, 1934.

Puget Sound Academy of Ophthalmology and Oto-Laryngology

President: Dr. Francis A. Brugman, 509 Olive St., Seattle.
Secretary-Treasurer: Dr. Noble Dick, 1115 Terry Ave., Seattle.
Time: 8 p. m., third Tuesday of each month, except July and August.

Rock River Valley Eye, Ear, Nose and Throat Society

President: Dr. Harry Shearer, 405 E. Grand Ave., Beloit, Wis.
Secretary-Treasurer: Dr. W. S. Beyer, 1101 Talcott Bldg., Rockford, Ill.
Place: Rockford, Ill., Janesville or Beloit, Wis. Time: Third Tuesday of each month.

Saginaw Valley Academy of Ophthalmology and Otolaryngology

President: Dr. A. R. McKinney, 703 Second National Bank Bldg., Saginaw, Mich.
Secretary-Treasurer: Dr. W. K. Slack, 308 Eddy Bldg., Saginaw, Mich.
Place: Saginaw, Mich., or Bay City, Mich. Time: First Friday of each month, except July and August.

Sioux Valley Eye and Ear Academy

President: Dr. John B. Gregg, 101 S. Main St., Sioux Falls, S. Dakota.
Secretary-Treasurer: Dr. F. H. Roost, 420 Trimble Bldg., Sioux City, Iowa.

Southern Medical Association, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Dunbar Roy, 113 N. Pryor St., Atlanta, Ga.
Secretary: Dr. William A. Wagner, 914 American Bank Bldg., New Orleans.
Place: New Orleans. Time: 1934.

Southwestern Michigan Triological Society

President: Dr. Carl Snapp, 604 Medical Arts Bldg., Grand Rapids.
Secretary-Treasurer: Dr. Lee O. Grant, 420 Medical Arts Bldg., Grand Rapids.
Time: Third Thursday of alternate months.

Western Pennsylvania Eye, Ear, Nose and Throat Society

President: Dr. O. G. A. Barker, 532 Main St., Johnstown.
Secretary-Treasurer: Dr. C. W. Beals, Weber Bldg., DuBois.
Time: Third Thursday of October and May.

STATE

Colorado Ophthalmological Society

President: A presiding officer is selected for each meeting alternately until all members have served.
Secretary: Dr. Edna M. Reynolds, 920 Republic Bldg., Denver.
Place: Capitol Life Building, Denver. Time: 7:30 p. m., third Saturday of the month, October to April, inclusive.

Connecticut State Medical Society, Section on Eye, Ear, Nose and Throat

President: Dr. Edward J. Whalen, 750 Main St., Hartford.
Secretary-Treasurer: Dr. Walter L. Hogan, 750 Main St., Hartford.
Time: May, November.

Eye, Ear, Nose and Throat Club of Georgia

President: Dr. W. R. Bedingfield, 753 Broad St., Augusta.
Secretary-Treasurer: Dr. J. A. Smith, 700 Spring St., Macon.

Indiana Academy of Ophthalmology and Oto-Laryngology

President: Dr. J. Kent Leasure, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Fred Overman, 611 Hume-Mansure Bldg., Indianapolis.
Place: Richmond. Time: Dec. 12, 1934.

Iowa Academy of Ophthalmology and Oto-Laryngology

President: Dr. W. F. Boiler, 105 E. Iowa Ave., Iowa City.
Secretary-Treasurer: Dr. Lawrence A. Taylor, 118 E. Second St., Ottumwa.

Michigan State Medical Society, Section of Ophthalmology and Otolaryngology

Chairman: Dr. Peter T. Grant, 148 Monroe Ave., Grand Rapids.
Secretary: Dr. Ralph B. Fast, 1410 Bank of Kalamazoo Bldg., Kalamazoo.
Place: Battle Creek. Time: 1934.

Minnesota Academy of Ophthalmology and Oto-Laryngology

President: Gordon B. New, Mayo Clinic, Rochester.
Secretary-Treasurer: Dr. Walter E. Camp, Medical Arts Bldg., Minneapolis.
Time: Second Friday of each month from October to May.

Montana Academy of Oto-Ophthalmology

President: Dr. Lawrence G. Dunlap, 101 Main St., Anaconda.
Secretary: Dr. A. W. Morse, Phoenix Bldg., Butte.

New Jersey State Medical Society, Section on Ophthalmology, Otology and Rhinolaryngology

Chairman: Dr. S. T. Hubbard, 294 State St., Hackensack.
Secretary: Dr. G. S. Laird, 125 Central Ave., Westfield.
Place: Atlantic City. Time: June, 1934.

New York State Medical Society, Eye, Ear, Nose and Throat Section

Chairman: Dr. Frank M. Sulzman, 1831 Fifth Ave., Troy.
Secretary: Dr. Webb W. Weeks, 20 E. 53rd St., New York.

North Dakota Academy of Ophthalmology and Oto-Laryngology

President: Dr. H. O. Ruud, 318 De Mers Ave., Grand Forks.
Secretary-Treasurer: Dr. F. L. Wicks, 516 Fourth St., Valley City.
Place: Valley City. Time: 1934.

OREGON ACADEMY OF OPHTHALMOLOGY AND OTO-LARYNGOLOGY
President: Dr. A. B. Dykman, Medical Dental Bldg., Portland.
Secretary-Treasurer: Dr. Andrew J. Browning, 418 Mayer Bldg., Portland.
Place: Good Samaritan Hospital. Time: Third Tuesday of each month.

RHODE ISLAND OPHTHALMOLOGICAL AND OTOLOGICAL SOCIETY
President: Dr. Robert C. O'Neil, 105 Waterman St., Providence.
Secretary and Treasurer: Dr. Gordon J. McCurdy, 122 Waterman St., Providence.
Place: Rhode Island Medical Library. Time: 8:30 p. m., second Thursday in October, December, February and April.

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. Walter Bristow, Medical Bldg., Columbia.
Secretary: Dr. J. W. Jervey, Jr., 101 Church St., Greenville.

TENNESSEE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY
President: Dr. C. D. Blassingame, 899 Madison Ave., Memphis.
Secretary-Treasurer: Dr. Ralph O. Rychener, 130 Madison Ave., Memphis.

TEXAS OPHTHALMOLOGICAL AND OTO-LARYNGOLOGICAL SOCIETY
President: Dr. C. C. Cody, 1304 Walker Ave., Houston.
Secretary: Dr. O. M. Marchman, 1719 Pacific Ave., Dallas.

UTAH OPHTHALMOLOGICAL SOCIETY
President: Dr. F. R. Slopanskey, 9 Exchange Pl., Salt Lake City.
Secretary-Treasurer: Dr. T. F. Welsh, 920 Boston Bldg., Salt Lake City.
Time: Third Monday of each month.

VIRGINIA SOCIETY OF OTO-LARYNGOLOGY AND OPHTHALMOLOGY
President: Dr. H. G. Preston, 307 Professional Bldg., Harrisonburg.
Secretary: Dr. Edwin W. Burton, 104 E. Market St., Charlottesville.

WEST VIRGINIA STATE MEDICAL ASSOCIATION, EYE, EAR, NOSE AND THROAT SECTION
President: Dr. Harry V. Thomas, 95 Fairmont Ave., Fairmont.
Secretary: Dr. W. F. Beckner, 1050 Fifth Ave., Huntington.

LOCAL

ACADEMY OF MEDICINE OF NORTHERN NEW JERSEY, SECTION ON EYE, EAR, NOSE AND THROAT
Chairman: Dr. Browne Morgan, 260 Liberty St., Bloomfield.
Secretary: Dr. A. Russell Sherman, 671 Broad St., Newark.
Place: 91 Lincoln Park South, Newark. Time: 8:45 p. m., second Monday of each month.

ACADEMY OF MEDICINE OF TORONTO, SECTION OF OPHTHALMOLOGY
Chairman: Dr. Colin Campbell, 170 St. George St., Toronto.
Secretary: Dr. H. M. Macrae, 170 St. George St., Toronto.
Time: First Monday of winter months.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY
President: Dr. W. L. McDougall, 50 Whitehall St., Atlanta, Ga.
Secretary: Dr. William O. Martin, Jr., 384 Peachtree St., Atlanta, Ga.
Place: Academy of Medicine, 38 Prescott St., N. E. Time: 8 p. m., last Thursday of each month.

BALTIMORE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY
Chairman: Dr. Benjamin Rones, Johns Hopkins Hospital, Baltimore.
Secretary: Dr. William M. Rowland, 906 Cathedral St., Baltimore.
Place: Medical and Chirurgical Faculty, 1211 Cathedral St. Time: 8:30 p. m., fourth Thursday of each month from October to May.

Brooklyn Ophthalmological Society

President: Dr. W. F. C. Steinbugler, 515 Ocean Ave., Brooklyn.
Secretary: Dr. Walter V. Moore, 1 Nevins St., Brooklyn.
Place: Kings County Medical Society Bldg., 1313 Bedford Ave. Time: Third Thursday in February, April, May, October and December.

Buffalo Ophthalmologic Club

President: Dr. H. H. Glosser, 448 Franklin St., Buffalo.
Secretary-Treasurer: Dr. I. J. Koenig, 40 North St., Buffalo.

Chicago Ophthalmological Society

President: Dr. Dwight C. Orcutt, 30 N. Michigan Ave., Chicago.
Secretary: Dr. Richard C. Gamble, 30 N. Michigan Ave., Chicago.
Place: Medinah Michigan Avenue Club, 505 N. Michigan Ave. Time: Third Monday of each month from October to May.

Cleveland Academy of Medicine, Section of Ophthalmology and Otolaryngology

Chairman: Dr. A. L. Stotter, 1148 Euclid Ave., Cleveland.
Secretary: Dr. James T. Collins, 1105 Medical Arts Bldg., Cleveland.
Place: Winton Hotel. Time: Fourth Friday of each month.

Cleveland Ophthalmological Club

Chairman: Dr. W. P. Chamberlain, 7405 Detroit Ave., Cleveland.
Secretary: Dr. A. D. Ruedemann, Cleveland Clinic, Cleveland.

College of Physicians, Philadelphia, Section on Ophthalmology

Chairman: Dr. John Milton Griscom, 2213 Walnut St., Philadelphia.
Clerk: Dr. Alexander G. Fewell, 1924 Pine St., Philadelphia.
Time: Third Thursday of every month from October to April, inclusive.

Columbus Ophthalmological and Oto-Laryngological Society

Chairman: Dr. Albert D. Frost, 681 E. Broad St., Columbus, Ohio.
Secretary-Treasurer: Dr. M. Goldberg, 328 E. State St., Columbus, Ohio.
Time: First Monday of each month.

Dallas Academy of Ophthalmology and Oto-Laryngology

President: Dr. T. S. Love, 1719 Pacific Ave., Dallas, Texas.
Secretary: Dr. Daniel Brannin, 1719 Pacific Ave., Dallas, Texas.
Place: Dallas Athletic Club. Time: 6:30 p. m., first Tuesday of each month from October to June. The November, January and March meetings are devoted to clinical work.

Des Moines Academy of Ophthalmology and Otolaryngology

President: Dr. C. C. Walker, 410 6th Ave., Des Moines, Iowa.
Secretary-Treasurer: Dr. Grace Doane, 614 Bankers Trust Bldg., Des Moines, Iowa.
Time: 7:45 p. m., third Monday of every month, September to May.

Detroit Ophthalmological Club

Chairman: Members rotate alphabetically.
Secretary: Dr. Raymond S. Goux, David Whitney Bldg., Detroit.
Time: 6:30 p. m., first Wednesday of each month.

Eastern New York Eye, Ear, Nose and Throat Association

President: Dr. Edward B. O'Keefe, 901 State St., Schenectady.
Secretary-Treasurer: Dr. W. C. Mott, 118 Washington Ave., Albany.
Time: Third Wednesday in October, November, March, April, May and June.

Fort Worth Eye, Ear, Nose and Throat Society

President: Dr. E. L. Howard, 600 W. 10th St., Fort Worth, Texas.
Secretary-Treasurer: Dr. Van D. Rathgeber, 1012 Medical Arts Bldg., Fort Worth.
Place: Medical Hall, Medical Arts Bldg. Time: 7:30 p. m., first Friday of each month except July and August.

Grand Rapids Eye, Ear, Nose and Throat Society

President: Dr. Carl F. Snapp, 26 Sheldon Ave., Grand Rapids, Mich.
Secretary-Treasurer: Dr. Walter W. Oliver, 320 Medical Arts Bldg., Grand Rapids, Mich.
Place: Various local hospitals. Time: Third Thursday of alternating months, September to May.

Houston Academy of Medicine, Eye, Ear, Nose and Throat Section

President: Dr. E. L. Goar, 1304 Walker Ave., Houston, Texas.
Secretary: Dr. J. C. Dickson, 622 Medical Arts Bldg., Houston, Texas.
Place: Medical Arts Bldg., Harris County Medical Society Rooms. Time: 8 p. m., second Thursday of each month from September to June.

Indianapolis Ophthalmological and Otolaryngological Society

President: Dr. D. A. Bartley, 23 E. Ohio St., Indianapolis.
Secretary: Dr. Kenneth L. Craft, 23 E. Ohio St., Indianapolis.
Place: University Club. Time: 6:30 p. m., second Thursday of each month from October to May.

Kansas City Society of Ophthalmology and Oto-Laryngology

President: Dr. M. B. Simpson, 1103 Grand Ave., Kansas City, Mo.
Secretary: Dr. Harold B. Hedrick, 618 Professional Bldg., Kansas City, Mo.
Time: Third Thursday of each month from September to May. The November, January and March meetings are devoted to clinical work.

Long Beach Eye, Ear, Nose and Throat Society

Chairman: Dr. Russell T. Uhls, 110 Pine Ave., Long Beach, Calif.
Secretary-Treasurer: Dr. Kenneth C. Brandenburg, 707 Security Bldg., Long Beach, Calif.
Place: Professional Bldg. Time: Last Wednesday of each month from October to May.

Los Angeles Society of Ophthalmology and Oto-Laryngology

President: Dr. F. H. Brandt, 1136 W. Sixth St., Los Angeles.
Secretary-Treasurer: Dr. Pierre Viole, 1930 Wilshire Blvd., Los Angeles.
Place: Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd. Time: 6:30 p. m., fourth Monday of each month from September to May, inclusive.

Louisville Eye, Ear, Nose and Throat Society

President: Dr. Charles Dwight Townes, 332 W. Broadway, Louisville, Ky.
Secretary-Treasurer: Dr. J. S. Bumgardner, 1103 Heyburn Bldg., Louisville, Ky.
Place: French Village. Time: Second Thursday of each month, September to May, inclusive, at 6:30 p. m.

Medical Society of the District of Columbia, Section of Ophthalmology and Oto-Laryngology

Chairman: Dr. George B. Trible, 1801 I St., Washington.
Secretary: Dr. Harry F. Davies, 1835 I St., Washington.
Place: 1718 M St., N. W. Time: 8 p. m., third Friday of each month from October to April, inclusive.

Memphis Society of Ophthalmology and Otolaryngology

Chairman: Each member, in alphabetical order.
Secretary: Dr. R. O. Rychener, Exchange Bldg., Memphis, Tenn.
Place: Eye Clinic of Memphis Eye, Ear, Nose and Throat Hospital. Time: 8 p. m., second Tuesday of each month.

Milwaukee Oto-Ophthalmic Society

Secretary-Treasurer: Dr. Herbert G. Schmidt, 238 W. Wisconsin Ave., Milwaukee.
President: Dr. O. P. Schoofs, 324 E. Wisconsin Ave., Milwaukee.
Place: University Club. Time: 6:30 p. m., fourth Tuesday of each month.

Montreal Ophthalmological Society

President: Dr. J. A. MacMillan, 1410 Stanley St., Montreal, Canada.
Secretary: Dr. A. L. Guertin, 410 Sherbrooke St., West, Montreal, Canada.
Time: Second Thursday of October, December, February and April.

Nashville Academy of Ophthalmology and Otolaryngology

Chairman: Dr. Frederick E. Hasty, 119, 7th Ave. N., Nashville, Tenn.
Secretary-Treasurer: Dr. Kate Savage Zerfoss, 803 Medical Arts Bldg., Nashville, Tenn.
Place: St. Thomas Hospital. Time: 8 p. m., third Monday of each month from October to June.

New Orleans Ophthalmological and Otolaryngological Society

President: Dr. M. F. Meyer, Canal Bank Bldg., New Orleans.
Secretary-Treasurer: Dr. R. H. Fisher, Chess and Checker Club, New Orleans.
Place: Eye, Ear, Nose and Throat Hospital. Time: Third Thursday of each month from October to June.

New York Academy of Medicine, Section of Ophthalmology

Chairman: Dr. Webb William Weeks, 20 E. 53rd St., New York.
Secretary: Dr. Guernsey Frey, 121 E. 60th St., New York.
Time: 8:30 p. m., third Monday of every month from October to May, inclusive.

Omaha and Council Bluffs Ophthalmological and Oto-Laryngological Society

President: Dr. Abbott M. Dean, 532, 1st Ave., Council Bluffs, Iowa.
Secretary-Treasurer: Dr. Robert H. Rasgorshek, 425 Aquila Court, Omaha.
Place: Omaha Club, 20th and Douglas Sts., Omaha. Time: 6 p. m., dinner; 7 p. m., program; third Wednesday of each month from October to May.

Philadelphia County Medical Society, Eye Section

Chairman: Dr. Charles R. Heed, 1205 Spruce St., Philadelphia.
Secretary: Dr. Sidney L. Olsho, 235 S. 15th St., Philadelphia.
Time: Second Tuesday of each month from October to May.

Pittsburgh Ophthalmological Society

President: Dr. Edward B. Heckel, Jenkins Arcade Bldg., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: Pittsburgh Academy of Medicine Bldg. Time: Fourth Monday of each month, except June, July, August and September.

Pittsburgh Slit-Lamp Society

President: Dr. W. W. Blair, 121 University Pl., Pittsburgh.
Secretary: Dr. George H. Shuman, Park Bldg., Pittsburgh.
Place: 121 University Pl. Time: Second Monday evening of every month, except June, July, August and September.

Richmond Ophthalmological and Oto-Laryngological Society

President: Dr. F. H. Lee, 501 E. Franklin St., Richmond, Va.
Secretary: Dr. William R. Weisiger, Medical Arts Bldg., Richmond, Va.
Place: Westmoreland Club. Time: 6 p. m., second Monday of each month from October to May.

Rochester Eye, Ear, Nose and Throat Society

President: Dr. E. W. O'Brien, 5 N. Goodman St., Rochester, N. Y.
Secretary-Treasurer: Dr. W. D. Edwards, 389 Monroe Ave., Rochester, N. Y.
Place: Rochester Medical Association, 113 Prince St. Time: 8 p. m., third Monday of each month from October to May.

St. Louis Ophthalmic Society

President: Dr. E. C. Spitze, 234 Collinsville Ave., East St. Louis, Ill.
Secretary: Dr. Carl T. Eber, 1006 Carleton Bldg., St. Louis.
Place: Oscar Johnson Institute. Time: Clinical meeting 5:30 p. m., dinner and scientific meeting 6:30 p. m., fourth Friday of each month from October to April, inclusive, except December.

San Antonio Ophthalmolo-Oto-Laryngological Society

President: Dr. Robert E. Parrish, 712 Medical Arts Bldg., San Antonio, Texas.
Secretary-Treasurer: Dr. E. King Gill, 323 Medical Arts Bldg., San Antonio, Texas.
Place: Bexar County Medical Library. Time: '8 p. m., first Tuesday of each month from October to May.

San Francisco County Medical Society, Section on Eye, Ear, Nose and Throat

Chairman: Dr. Robert S. Irvine, 490 Post St., San Francisco.
Secretary: Dr. Frank H. Rodin, 490 Post St., San Francisco.
Place: Society's Building, 2180 Washington St., San Francisco.
Time: Fourth Tuesday of every month except May, June, July and December.

Shreveport Eye, Ear, Nose and Throat Society

President: Dr. W. L. Atkins, 940 Margaret Pl., Shreveport, La.
Secretary-Treasurer: Dr. Leon F. Gray, 1214 Slattery Bldg., Shreveport, La.
Place: 1240 Texas Ave. Time: 8:15 p. m., fourth Tuesday of every month except June, July and December.

Spokane Academy of Ophthalmology and Oto-Laryngology

President: Dr. Marc Anthony, 422 Riverside Ave., Spokane, Wash.
Secretary: Dr. Raymond A. Lower, 1455 Paulsen Medical and Dental Bldg., Spokane, Wash.
Place: Paulsen Medical and Dental Library. Time: 8 p. m., fourth Tuesday of each month except June, July and August.

Syracuse Eye, Ear, Nose and Throat Society

President: Dr. H. O. Brust, 820 S. Crouse Ave., Syracuse, N. Y.
Secretary-Treasurer: Dr. W. J. Werfelmann, 109 S. Warren St., Syracuse, N. Y.
Place: University Club. Time: Second Friday of each month except June, July and August.

Tulsa Academy of Ophthalmology and Otolaryngology

President: Dr. Roy Dunlap, Medical Arts Bldg., Tulsa, Okla.
Secretary-Treasurer: Dr. Marvin D. Henley, Medical Arts Bldg., Tulsa, Okla.
Place: Medical Arts Bldg. Time: Monthly.

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